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CUSHING'S DISEASE—PITUITARY BASOPHILISM

CALDWELL LECTURE, 1947*

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BOSTON, MASSACHUSETTS

INTRODUCTORY REMARKS

By Arthur C. Christie, M.D.

The Caldwell Lecture for which we are assembled here tonight was established by the American Roentgen Ray Society in the year 1920, through the initiative of Dr. James T. Case, who was then President of the Society. It is a great privilege to have Dr. Case here tonight.

The Lecture has become increasingly important through the years, not only because of our increasing realization of the greatness of the man for whom it was founded, but because of the growing prestige of the oldest of American radiological societies and the now imposing list of illustrious men who have from year to year delivered it.

Eugene Caldwell has now been dead for thirty years and few remain who knew him intimately. Although I was much younger than he, it was my privilege to be in somewhat intimate contact with him from time to time during the last year of his life. He was working intensively to perfect a stereofluoroscope, hoping that it could be used in the first World War in which we were then engaged, and knowing that his own time was rapidly running out.

He impressed me then as a great man. He had many of the qualities that contribute to greatness of mind and soul, but there was one dominant characteristic which stands out vividly in my memory of him to this day. It was the courage with which he faced the hard problems and circumstances of his waning days. I do not mean the kind of courage that most of us have, which can rise to meet some great crisis, or even catastrophe, but the kind of courage which carries on the ordinary affairs of life in spite of grinding physical pain, and in the knowledge that death awaits to end all activity in a very short time. Under such circumstances I remember Caldwell, pursuing his work with enthusiasm, meeting difficulties with good nature and a constant quiet humor.

There was what I can only describe as a radiant quality about the man that remains with me to this day as my most vivid memory of his personality. Aldous Huxley has aptly said that experience is not what happens to a man; it is what a man does with what happens to him. It is the prize he receives for the way in which he deals with the accidents of existence. Caldwell dealt with the circumstances that surrounded him in his day in such a way that he has left a great heritage to us, his successors in radiology, and we commemorate his achievements again this year in the Caldwell Lecture.

Merrill Sosman, who is to deliver the Lec-

* Delivered before the Forty-eighth Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 16–19, 1947.

ture tonight, is a worthy successor to this pioneer in radiology. He has had an important part, by original contributions and as a teacher and leader, in the progress of radiology during the present generation. His career has in it encouragement for every young American radiologist who may be just starting his life's work.

He was born and reared in a small town of the Middle West and graduated from a typical middle western university, albeit one of the best. I remember in my own graduating class there were those who graduated *cum laude* (with praise), those who graduated *magna cum laude* (with great praise), and one who even graduated *summa cum laude* (with the highest praise). A classmate, who remembered his Latin better than most, said that the rest of us graduated *mirabile dictu* (wonderful to tell).

I do not know in which of these categories Dr. Sosman found himself at graduation, but his career may well be characterized by the words "mirabile dictu," for it is really a marvelous story to tell. I wish I had time to tell it in detail but I must be very brief in order that you may come unwearied to his Lecture.

After graduation at the University of Wisconsin he entered the Johns Hopkins Medical School and graduated in medicine just at the beginning of the first World War and soon found himself in the Medical Corps of the Army. It was there that he became interested in radiology, having become, like the present speaker, a radiologist "by order of the Surgeon General."

When the war was over, he continued his training under one of the greatest teachers of radiology that America has produced, George W. Holmes. From his course of training he went directly to the Peter Bent Brigham Hospital to be associated as radiologist with Harvey Cushing. There his entire life's work up to the present moment has been done. How productive, how even brilliant that work has been, I need not relate to an audience of radiologists. He has there exemplified all of those qualities which contribute to the making of a great radiologist and a great physician.

He comes tonight, at the zenith of his career, to acquaint you with some of the results of his work.

Dr. Sosman, the American Roentgen Ray Society has formerly honored you by electing you President of the Society. Tonight it gives you one of the highest honors that it can bestow by naming you the Caldwell Lecturer. In bestowing these and other honors upon you, the Society feels not only that it has honored you, but that you do honor to the Society by your entire career.

Ladies and gentlemen, it is my great personal honor and privilege to present to you the Caldwell Lecturer, Dr. Merrill C. Sosman, Professor of Radiology at Harvard University Medical College. Dr. Sosman!

CALDWELL LECTURE

PROLEGOMENON

To be chosen to give the Caldwell Lecture is not only a great honor, but it is also a great responsibility. Named for Eugene Wilson Caldwell, one of the pioneers and martyrs in our specialty, and initiated by Dr. James T. Case in 1920, this is the twenty-sixth of the series to be given before this Society. Eleven of these "lectures" have been given by roentgenologists, seven of them former presidents of this Society, four from abroad. Nine have been given by medical but non-roentgenological colleagues, and five by physicists. The subjects of the lectures have been about equally divided between general or abstract topics and specific or concrete talks on clinical matters of particular interest to the speaker, and, I hope, to his audience. Just twenty-three years ago (1924) we met at Swampscott, Massachusetts, and Dr. Gösta Forssell gave the Caldwell Lecture on "Experiences on the Permanancy of the Radium Cure in Malignant Tumors." That year I became a member of this Society. Dr. George W. Holmes sponsored me for membership. Dr. William Duane allowed me to observe him at work in his laboratory at Harvard, and Dr. Harvey Cushing and the whole staff at the Brigham Hospital also aided in the continuous post-graduate education which I have enjoyed for the past twenty-three years. All of us owe our intellectual debts to our predecessors upon whose work we build, and it is well to pause at least once a year to acknowledge that debt as we do tonight.*

* The accepted meaning of "lecture" in the English universities is "a rehearsal of a lesson." (Webster.) This is a lesson I have learned (and am still learning) since 1930.

Eugene Wilson Caldwell was a symbol of his age and of his period. Born in Savannah, Missouri, he grew up and went to school during the time when electricity was developing and growing, with new uses being found for it almost daily. His training at school and college was in electrical engineering and his early work was with submarine telephones, wireless, and other electrical specialties. Acquiring a photographer's outfit in 1897 which included a roentgen-ray machine, he became interested in our specialty and from then on spent most of his time doing roentgen-ray work and improving roentgen-ray apparatus and tubes in his own workshop. Realizing the necessity of having an M. D. degree in order to practice roentgenology, in 1901 he entered Bellevue Medical School, where he was already on the faculty as Director of the Edward N. Gibbs Memorial X-ray Laboratory. He had opened and equipped the X-ray Laboratory at Bellevue Hospital in 1900 with the help of the Gibbs Foundation, and was in charge of the department until he resigned in 1908. His whole life history reads very much like that of Walter Dodd, in Boston, and to both of them, who died of X-ray cancer, one in 1916, the other in 1918, as well as to many others of their generation, these lectures are dedicated. Caldwell's published works dealt mostly with clinical subjects, but his practical improvements in machines, tubes, and equipment equalled his literary contributions in importance. As early as 1903, Caldwell and Pusey published a book "The Practical Application of the Roentgen Ray in Therapeutics and Diagnosis," but Caldwell is best remembered in our specialty for his work "Skiagraphy of the Sinuses," in which he advocated the frontonasal projection now known as the "Caldwell position." He also reported a study on the epiphyseal development in children, trying to correlate it with their mental development, but concluded that there was no constant relationship. Of particular interest, in view of the subject tonight, was his study of the sella turcica in epileptic patients, a study with essentially negative results. Other subjects, indicative of his wide range of interest, were articles on renal calculi, gallbladder examinations, and the stereoscopic fluoroscope, which was nearing perfection when he died in 1918.

The title of this evening's talk could just as well have been "Pleasure and Satisfaction in Roentgenology" rather than "Cushing's Disease—Pituitary Basophilism." It has been a constant source of pleasure and satisfaction to be in a teaching hospital where research and investigation are considered a privilege as well as a duty and which are, *ipso facto*, a part of one's job. To take part in some of these investigations, even the small and insignificant part I have taken, gives one a thrill and a satisfaction which no other work can do.

The work of a roentgenologist in a large hospital is necessarily a reflection of the interests of his staff. If there is clinical material unusual in number or type, he should seize the opportunity to develop special competence along those lines and contribute his experiences to his fellow workers who do not have access to the same rich vein of material. It is only the decent thing to do and no one deserves praise or commendation for doing his share toward solving some of the many unsolved problems in medicine.

Pituitary basophilism was suggested in 1930 by Teel and Cushing*[62] as a possible addition to the well known hyperpituitary state of acromegaly and the hypopituitary state of the patients with chromophobe pituitary adenomas. It remained for one of Cushing's students, H. M. Teel,[61] first to observe such a patient and to predict (in 1931) the basophilic adenoma which was subsequently found at autopsy.

* Cushing was writing a paper with Teel on the growth hormone and the sex hormone of the pituitary gland. In reviewing the literature, he found a patient reported by Raab[55] and Kraus[41] in 1924 whose clinical condition closely simulated that of a patient then in the hospital (our case 1, E. F.). Cushing predicted a basophil adenoma in his patient, as had been found at autopsy in Raab's patient, but he had to wait until 1935 for postmortem verification.

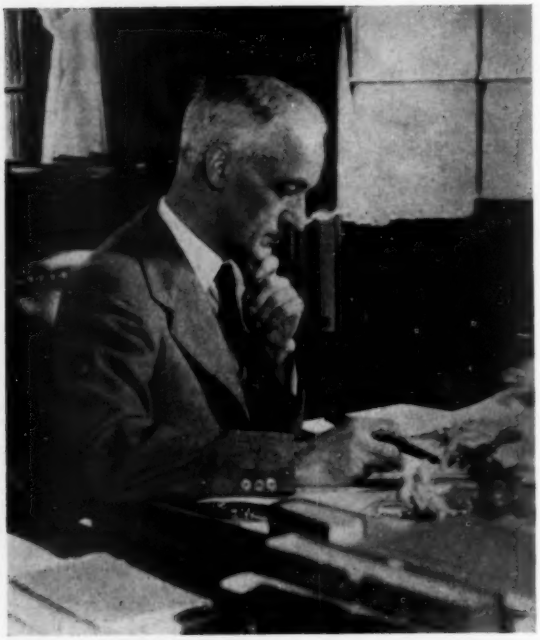

Fig. 1. Harvey Cushing, 1869–1939. (Photograph by Walter Boyd, M.D.)

With characteristic energy, Cushing collected and assembled all the material then available (only 12 cases, including 2 of his own patients), with an enormous amount of collateral and supporting evidence, and described the syndrome of "pituitary basophilism." The assembled material was first presented formally before the New York Neurological Society on January 5, 1932. Also with characteristic facility, he presented the same material next before the Harvard Medical Society, January 20, 1932, then as the Alpha Omega Alpha Lecture at New Haven* on February 24, 1932, and again to the Johns Hopkins Medical Society on February 29, 1932. The complete article was first published in the *Bulletin of the Johns Hopkins Hospital* in March, 1932,[15] and immediately aroused great interest as well as a storm of criticism and dissension over his deductions. Subsequent amplifications of this original concept were published in the *Journal of the American Medical Association* in July, 1932,[16] adding 4 cases, including 2 new Brigham Hospital patients whose progress I shall report on tonight. His third paper on the subject was given as the Harvey Lecture in 1933[17], entitled "Dyspituitarism; Twenty Years Later." This was his second Harvey Lecture and one in which he contrasted the knowledge of the pituitary gland in 1912, at the time of his first Harvey Lecture, with the knowledge twenty years later.

It is unnecessary, I am sure, to remind you of what an important part Harvey Cushing (Fig. 1) played in the development of knowledge concerning the pituitary body.† It should be interesting, however, to review briefly some of the historical land-

* The room which was readied for Cushing's talk was filled to overflowing before the address was begun. The meeting was adjourned to a larger auditorium, but unfortunately no lantern or screen was available, and Cushing had to describe his patients rather than project their photographs on the screen. But he rose to the occasion and charmed his audience with his masterly word pictures. (Fulton[21].)

† When I joined the Brigham Staff in 1922, I was gently criticized by an Important Person for speaking of the "pituitary gland," and was told that there was no reliable evidence that the anterior lobe secreted anything which would justify calling it a gland. Hence the term "pituitary body."

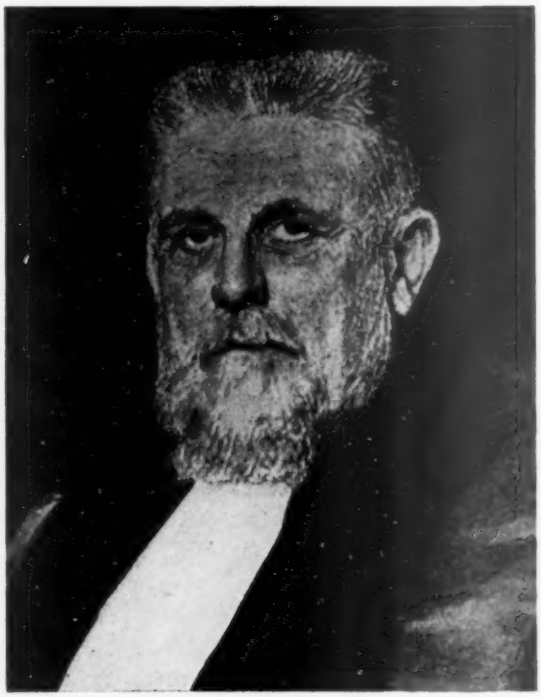

Fig. 2. Pierre Marie, 1853–1940.

marks in the recognition of pituitary tumors and their amazing effects upon the human organism due to changes in its secretions.

The great French neurologist, Pierre Marie (Fig. 2), is universally credited with the first description[47] in 1886 of acromegaly and its relation to a tumor of the hypophysis, but the condition had been described previously (in 1884) by Fritsche and Klebs.[28] Marie reported 2 cases of his own and collected 5 cases from the literature (Fig. 3). Only one patient (Henrot's) had been autopsied, a large pituitary tumor having been found, but Marie did not attribute the condition to the pituitary gland. His main thesis was the differentiation of this condition, which he named "acromegalie," from Paget's disease, leontiasis ossea, and myxedema.

In 1887 Minkowski[49] reported one case and called attention to the case reported in 1884 by Fritsche and Klebs, who had found at autopsy enlargement of both thymus

SUR DEUX CAS D'ACROMÉGALIE

HYPERTROPHIE SINGULIÈRE NON CONGENITALE DES EXTRÉMITÉS SUPÉRIEURES, INFÉRIEURES ET CÉPHALIQUE

Par Pierre MARIE

Chef de laboratoire adjoint à la Salpêtrière.

Les deux observations qui font l'objet de ce travail ont été recueillies dans le service de M. le Professeur Charcot ; elles ont trait à une affection qui n'a pas encore été isolée et décrite dans son ensemble ; cependant il semble bien qu'on soit là en présence d'une *entité morbide spéciale*, car chez tous les malades on retrouve d'une façon surprenante le même aspect, les mêmes caractères. Cette affection est-elle très rare ? nous l'ignorons ; pour notre part nous en avons observé deux cas simultanément, mais dans les recherches bibliographiques assez étendues auxquelles nous nous sommes livré nous n'avons pu en retrouver qu'un très petit nombre d'exemples [1].

Fig. 3. Marie's report of "Two Cases of Acromegaly."

and the pituitary gland. They had assumed the thymus to be the cause of the condition and explained the enlarged pituitary gland as part of the generalized overgrowth. Minkowski, however, attributed the acromegaly to the enlarged pituitary gland, reasoning by analogy with thyroid gland function and myxedema.

Marie considered the condition to be one of general nutritional disturbance, modified by local vascular supply and by inherent embryological tendencies.*

Fig. 4. Alfred Fröhlich, 1871–. (Photograph taken in 1945.)

It was fifteen years later that Fröhlich[29] (Fig. 4) published his case entitled "Pituitary tumor without acromegaly," and described the syndrome of a short fat, sexually undeveloped fifteen year old boy (Fig. 5). This is now known as "Fröhlich's syndrome." Unknown to him, Babinski[6] the French neurologist, had described a

* For interesting and complete historical details concerning the development of theories and knowledge about acromegaly see Davidoff.[18]

Originalartikel, Berichte aus Kliniken und Spitälern.

Aus der I. medicinischen Klinik des Herrn Hofrathes Prof. H. Nothnagel.

Ein Fall von Tumor der Hypophysis cerebri ohne Akromegalie.

Von **Dr. Alfred Fröhlich.***)

M. H.! Ich möchte mir erlauben, Ihnen einen Fall zu demonstrieren, den ich in dem von Herrn Professor v. Frankl-Hochwart geleiteten Nerven-Ambulatorium der Klinik des Herrn Hofrathes Nothnagel zu beobachten Gelegenheit hatte und der mir in mancherlei Hinsicht Bemerkenswertes darzubieten scheint.

FIG. 5. Fröhlich's report of "A Case of Tumor of the Hypophysis without Acromegaly."

similar proved case a year earlier, but the popularity of the Viennese School was such that Fröhlich's, rather than Babinski's name was attached to the syndrome.*

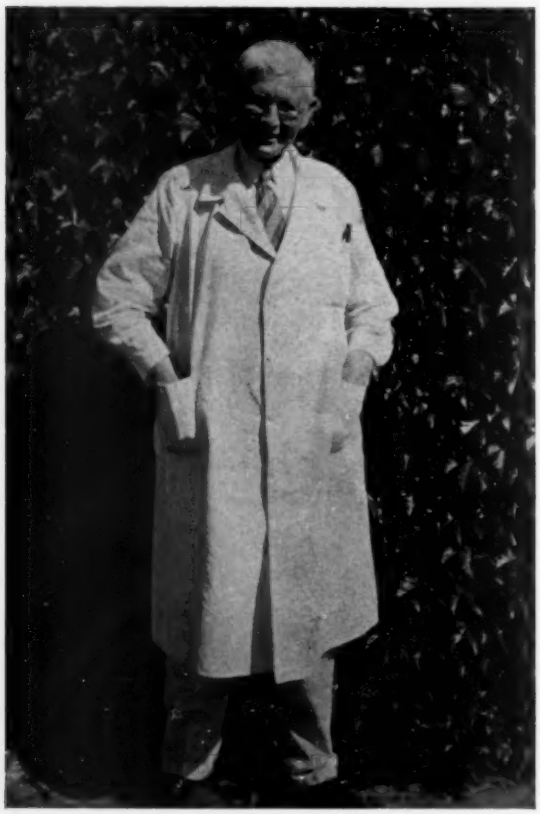

FIG. 6. Jacob Erdheim, 1874–1937. (Photograph taken in Vienna, 1931.)

* It is interesting to note here that Fröhlich persuaded the Viennese neurosurgeon, von Eiselsberg, to operate on his original patient, and a cystic tumor of this type was found; but too late to save the child's vision. The patient, however, was still alive when Fröhlich had to leave Vienna for this country in 1939. (Fulton.[22])

The next major contribution to our knowledge of tumors of the pituitary gland was by Jacob Erdheim (Fig. 6), the pathologist of Vienna, who showed that the tumors most apt to produce Fröhlich's syndrome were suprasellar in position, teratomatous in character, and often distorted the hypothalamus, which could explain the obesity. Erdheim made many important contributions to the histology of the pituitary gland and its tumors, and was the first to identify a basophilic adenoma in 1933.[22] It remained for Cushing to develop in 1909 the concept of hyperpituitary and hypopituitary states and his monograph "The Pituitary Body and Its Disorders" published in 1912[14] is still a veritable mine of information, and a prize to be acquired in secondhand book stores. Cushing was the pioneer in brain surgery as Caldwell was in roentgenology; both were indefatigable, always alert, and never satisfied with things as they were, ever striving for something better.

PITUITARY TUMORS IN GENERAL

But what do we know about these varied pituitary tumors? We know that there are two main types, the craniopharyngiomas and the adenomas. The former are the congenital cysts arising from a remnant of Rathke's pouch, usually suprasellar in position, frequently (70 per cent) containing enough calcium for the roentgenologist to recognize them on the roentgenograms.[48] They are found mostly in children, usually give rise to a hypopituitary state of adiposity, dwarfism, and sexual infantilism, as described by Fröhlich, and cause blindness if not relieved by operation. They do not respond to roentgen irradiation in my experience.

The pituitary adenomas are of three main types, classified by their histopathological appearance as (*a*) chromophobe, (*b*) acidophil and (*c*) basophil. About 70 per cent of the 338 verified adenomas in Cushing's series were of the chromophobe type, 20 per cent were acidophilic and 10 per cent were mixed (*d*) chromophobe and acidophil

cells. The basophilic adenomas are rare. Only two with the syndrome which I shall discuss later were verified by autopsy in Cushing's series.

(*a*) The chromophobe cells are presumably the mother cells of the pituitary gland. They are nongranular, poorly staining cells and have no known secretion or function in the human. Adenomas formed by this type of cell, therefore, are nonsecretory, and cause symptoms only by compressing neighboring structures, such as the normal cells of the pituitary gland, the optic chiasm and, if they extend beyond the sella, the third ventricle, temporal lobe or hypothalamus. Due to compression of the normal acidophil and basophil cells, their *endocrine* symptoms are those of hypopituitarism, such as amenorrhea or impotence, sluggishness, sensitivity to cold, and changes in bodily characteristics such as thin fine hair, thin dry skin, lack of perspiration and scanty axillary and pubic hair. Such tumors are rarely seen before puberty so that growth is not affected. Most of these adenomas make themselves evident between thirty and forty-five years of age, but the diagnosis may not be made before ten or fifteen years have passed.

The *neighborhood* symptoms are chiefly those of pressure on the hypothalamus leading to obesity and somnolence, and those due to pressure on the optic chiasm or nerves with resultant failure of vision. The characteristic finding is a bitemporal visual field defect. Failing vision, with or without headaches, is the most common reason for this type of patient to seek medical advice. These tumors responded to irradiation in 80 per cent of our series, often with prolonged remissions of symptoms and improvement or restoration of vision. An index of the value of roentgen treatment is the table from Henderson's[36] follow-up of all of Cushing's 338 confirmed adenomas, which show a much higher freedom from recurrence five years after operation in those patients who had postoperative irradiation (Table 6a). About 17 to 20 per cent of the chromophobe adenomas are cystic and do not respond well to irradiation alone, but postoperative irradiation is just as beneficial in this group, preventing or postponing recurrences, as in the solid adenomas.

(*b*) The acidophil adenomas are made up of coarsely granular cells which take the acid eosin stain well, and are actively secreting tumors. They occur only occasionally before growth has ceased,* but when they do, cause gigantism. More commonly they occur at thirty to forty years of age and acromegaly is the result. Rarely acromegaly is superimposed upon gigantism. In contrast to patients with chromophobe adenomas, these patients show the endocrine effects of *hyper*pituitarism, chiefly of the growth factor, which leads to gigantism in young persons or acromegaly in adults. Concurrently there is often an overproduction of other pituitary hormones such as the thyroid-stimulating one, certain gonadotropins and some as yet ill defined anti-insulin factor. The overgrowth is not limited to the skeleton, as many think, but involves all the viscera and probably all body tissues. Also in contrast to the *hypo*pituitary state in patients with chromophobe tumors, these acromegalics have coarse abundant hair, thick skin with excessive perspiration, increased basal metabolic rate, excessive menstruation or increased libido et potentio, decreased sugar tolerance, sometimes with glycosuria, and marked mental disturbances. The acromegalic patient is apt to be obstreperous, cantankerous, and difficult to handle, in contrast to the patient with a chromophobe adenoma who is apt to be placid, acquiescent and cooperative. Less than half of the patients with acidophil adenomas suffer from visual failure, but headache is often the presenting or outstanding complaint. These patients often complain of "waves" of acromegalism with accession and recession of their symptoms for no apparent cause. (The patients with basophilic tumors also have recurrent "waves" of activity which are poorly understood.)

* Only 6 in Cushing's series of 75 patients.

The roentgen examination of the skull in the acromegalic group shows, in addition to the coarse, heavy, thickened bones, enlarged nasal accessory sinuses, enlarged mandible, and an enlarged pituitary fossa in 80 per cent of the cases. Twenty per cent of the acromegalics in our series had normal sellae in contrast to the chromophobe group where only 7 per cent had normal sellae and 93 per cent had enlarged "ballooned" pituitary fossae. Calcification in or around these adenomas is rarely seen but when present is probably due to hemorrhage or to necrosis in the tumor.

The effects of irradiation are difficult to assay in the acidophil group due to the "waves" of acromegalism, and due to the fact that spontaneous remissions are common. Not infrequently the acromegalic patient after some years of activity spontaneously changes from a *hyper*pituitary state to one of *hypo*activity, but the acromegalic features do not regress. The headaches, often the most distressing symptom, were relieved by irradiation in 90 per cent of our patients, and we believe, although it is hard to prove, that the development of acromegaly (or gigantism) can be stopped by prompt and adequate irradiation. The most common causes of death in acromegalic patients, in order of frequency, are (1) diabetes, (2) heart failure, (3) hypertension and apoplexy, and (4) intracranial extension.

(*d*) The signs and symptoms in patients with "mixed" adenomas are usually the same in the beginning as those of acromegaly, but then the development of the classical acromegalic state stops and the patients present the more common features of hypopituitarism of the chromophobe type of adenoma. Cushing[15] called this "fugitive acromegaly" and explained it by assuming that the chromophobe cells outgrew the acidophil cells and, by compressing them, stopped their function. The subsequent course of the signs and symptoms is that of the chromophobe adenomas.

PITUITARY BASOPHILISM

(*c*) To return to the basophil adenomas or pituitary basophilism, we find in this syndrome the most bizarre and complex signs and symptoms of polyglandular dyscrasia. This syndrome is characterized by:

1. A peculiar plethoric painful adiposity of the face, neck and trunk, sparing the extremities.
2. Cyanosis of the face, neck, hands, and feet.
3. Amenorrhea in the female; loss of libido et potentio in the male.
4. Hypertrichosis in the female with decrease in normal hair.
5. Muscular weakness, irritability, drowsiness or depression.
6. Changes in the skin, which is thin, dry, mottled and scaly, often with pigmentation; easy bruisability, slow healing, and facial acne.
7. Polyphagia, polydipsia and polyuria.
8. Osteoporosis of spine, skull and pelvis.
9. Glycosuria or decreased sugar tolerance.

Any of these signs or symptoms may be absent in an individual patient, or delayed in appearance, and no one symptom or group of symptoms is essential in the syndrome. Less frequent in occurrence and sometimes absent from the syndrome are:

10. Vascular hypertension, sometimes fluctuating.
11. Polycythemia of mild degree.
12. Low basal metabolic rate in the majority, occasionally normal or even elevated.
13. Cardiac enlargement, usually present at autopsy, probably a result of the hypertensive state.
14. Arteriosclerosis, generalized.
15. Nephrolithiasis.

Different authors emphasize various features of the syndrome, but all agree that the condition is a true syndrome, of variable degree and intensity as to its individual features, and of hotly debated and unproved etiology at present. Cushing attributed the entire syndrome to the basophil adenoma which has been found in

most but not all of the autopsied cases. He acknowledged the close similarity to the signs and symptoms in some patients who had adrenal cortical tumors, and stressed the part played by the overactive adrenal glands, but attributed the initial etiological stimulus to the basophil cells of the pituitary gland in all instances. The storm of discussion and criticism which I mentioned earlier chiefly concerned the explanation of the syndrome as of pituitary rather than adrenal origin, and did not apply to the clinical features of the syndrome. Those who were in a position to know or to understand the syndrome quickly took sides in the controversy, and the endocrine field became divided into two opposing camps, the Pituitary Basophil army and the Adrenocortical forces. (There were Big Guns and Heavy Artillery in both camps and active bombardments ensued.)

Before going into the various solutions suggested for this controversial point it might be profitable to take stock of our present knowledge of the pituitary gland as it may be affected in Cushing's syndrome.

PITUITARY-ADRENAL MECHANISMS

When Cushing first described the signs and symptoms of what he then termed "pituitary basophilism" the knowledge of the endocrinology of the pituitary gland was just undergoing a tremendous, yet somewhat vague, expansion. While it was generally recognized then that the pituitary elaborated hormones which stimulated a great number of other endocrine glands and some of which directly affected metabolic processes, their nature, source and exact mode of action remained to be determined, for the most part, during the next two decades.

Today we know that the anterior lobe of the pituitary gland secretes, among others, a hormone known as the adrenocorticotropic hormone* which is thought to be formed by the basophilic cells.

* This moderately well characterized substance is known as ACTH for short.

Sensory stimuli acting through the central nervous system increase the secretion of the adrenocorticotropic hormone and so does adrenalin. The adrenalin in turn arises from the adrenal medulla through stimulation via the sympathetic nervous system whenever the organism is put into a state of alarm. The adrenocorticotropic hormone, carried to the adrenal cortex by the blood stream, profoundly affects the secretory cells of this vital gland. There is an immediate release of the cortical hormones and, if the stimulus is long continued, there follows a period of exhaustion; then an increase in the number of cells, a true hyperplasia, which is accompanied by an excessive secretion of adrenal cortical hormones. This series of physiological changes centering around both independent components of the adrenal gland constitutes a rather beautiful extension of the concepts of the late Walter Bradford Cannon, the eminent physiologist and early roentgen-ray pioneer, as to the important part the sympathetic system plays in the survival of the body in inclement environment. Fuller Albright[1] has pointed out the similarity of the endocrine disorders in Cushing's syndrome to those of the alarm reaction of Selye.[57]

To date the adrenocorticotropic hormone has not been shown to exert any metabolic effect on any known system in the absence of a responsive adrenal cortex. In the absence of the adrenocorticotropic hormone adrenal cortical function appears to be able to carry on to a slight degree, but is unable to respond to an alarming situation by an increase in its secretory activity, —i.e. it is adequate for the organism under basal conditions but fatally incompetent under even the mildest of stresses, such as infection.

From this brief summary it should be obvious that there exists a rather delicately balanced mechanism for the maintenance of "homeostasis," to use Cannon's convenient term, in which both the anterior pituitary and the adrenal cortex play their part. The existence of a functional adenoma

in either of the glands will lead to a pathological increase in their secretory activity. A malignant condition, no longer subject to any regulatory action, will produce even more striking effects.

What are the effects of adrenal cortical secretions? At least three functional types of hormone are known to arise from the adrenal cortex:

(1) Those which by preserving the sodium and water for the organism regulate the state of hydration, blood volume and blood pressure. The "salt hormone," deficient in Addison's disease, is present in some excess in most cases of Cushing's syndrome. Desoxycorticosterone, synthesized in 1937 and commercially available for practical treatment, is an example of this type of adrenal hormone.

(2) Those closely related to the male sex hormones of the testes which contribute to secondary male sex characteristics and tend to enhance the formation of muscle tissue at the expense of carbohydrate and fat formation. The "N" hormone or androgens are deficient in eunuchism and excessive in masculinizing tumors of the adrenal but not much elevated in Cushing's syndrome, as a rule. Androsterone, closely related to testosterone, which is available for therapeutic use, is a representative androgen.*

(3) A group of hormones known as the corticoids or "S" hormones which counteract the androgens by diverting body tissue into the metabolic pool for the ultimate formation of carbohydrate and fat at the expense of muscle and bone matrix. They tend to increase liver glycogen, blood sugar and body fat, depleting the muscle mass and leading to osteoporosis. They also regulate certain of the blood elements, leading to the destruction of lymphoid tissue,[21,24] decreasing circulating eosinophils and increasing the neutrophil count of the blood.[24,37] The "S" hormone or corticoids are greatly increased in cases of Cushing's syndrome, the urinary excretion being over ten times the normal, as a rule. Corticosterone and its derivatives are examples. None has been prepared in sufficient quantity for therapeutic use, but the whole extracts of adrenal cortex such as "cortin" contains these factors in variable amounts.

The relation of the adrenocorticotropic hormone to the excretion of these factors in man has recently been clarified by work done on the medical wards of the Peter Bent Brigham Hospital by Thorn and his collaborators. They found that the administration of adrenocorticotropic hormone to humans leads to nearly all of the metabolic changes which one might expect to obtain if all three cell types had been activated. Salt retention and corticoid-like activity were affected much more markedly than the production of androgens, a picture which closely simulated the findings in Cushing's syndrome, many of the metabolic changes of which were actually reproduced.[24]

With this somewhat oversimplified description of established mechanisms in mind, the variety of findings in Cushing's syndrome should appear somewhat less irrational.

PATHOLOGICAL PHYSIOLOGY

A basophilic adenoma *per se*, in the absence of increased adrenal cortical activity, could not be expected to give rise to any of the metabolic changes that lead to the development of Cushing's syndrome. The syndrome may be present in the absence of demonstrable adrenal cortical changes, which is only the reflection of our inability to demonstrate functional changes at postmortem. In fact, in rare cases such as the one reported by Freyberg *et al.*,[2] atrophy of the adrenal cortices may be found. This would represent a state of complete adrenal cortical exhaustion following continued stimulation by adrenocorticotropic hormone with a fulminating atrophy and death in an adrenal crisis with the anatomical findings of Cushing's syndrome still present elsewhere in the body.

* The 17-ketosteroids are breakdown products of the "N" hormones or androgens, and not necessarily of androgenic activity in this form.

Conversely, a basophilic adenoma need not necessarily be expected in every case of Cushing's syndrome since a primary adenoma of the adrenal cortex or a tumor of the primordial cells of the ovary such as an arrhenoblastoma may produce many of the characteristic changes.

Whether the initial stimulus to adrenal hyperplasia arises in the pituitary cells or in the cells of the adrenal cortex, the syndrome will still be characterized by the effects of the particular adrenal cortical hormones which are produced in excess. The syndrome will vary somewhat depending on which type of hormone predominates.

On the one hand, we have the adrenogenital syndrome in which excessive secretion of androgens leads to the characteristic increase in muscularity and male sex characteristics. The most marked cases represent primary adrenal tumors as a rule. On the other hand, we have true "Cushing's disease" in which excessive adrenocorticotropic hormone production leads to increased corticoid secretion producing the generalized tendency to loss of protoplasm, fat formation and diabetes. Between these extremes extends a veritable spectrum of abnormalities comprising Cushing's syndrome.

The next major contribution to our knowledge of pituitary basophilism was that of Crooke[12] in the pituitary camp, who described hyalinization of the basophil cells in the anterior lobe of the pituitary gland in Cushing's syndrome, both with and without adrenal tumors and basophil adenomas. This was accepted by many as the common denominator of the syndrome, but was explained by others, e.g., Severinghaus,[58] as a result of the condition (degeneration of the basophil cells) rather than the cause. It is interesting to note, in this respect, that the cells of the basophil adenomas, when present, usually do not have the hyaline change which is found in the other basophil cells. Crooke's findings were substantiated by Thompson and Eisenhardt[63] who reviewed the 94 cases reported in the literature up to 1940. They personally studied sections of the pituitary glands in 63 of the cases and found Crooke's changes in all but 5 of them, and added that these 5 were not clinically satisfactory instances of the syndrome.

Albright, Parson and Bloomberg[2] characterized the syndrome as one of hyperadrenocorticism with respect to the "S" or sugar hormone of the adrenal cortex, and clearly differentiated the adrenogenital syndrome from Cushing's syndrome. They agreed with Kessel [40] who in 1936 suggested that it was logical to designate the entire syndrome as "Cushing's syndrome," but to call those with proved basophilic adenomas and without adrenal tumors "Cushing's disease." Albright elaborated three successive theories (1941, 1942 and 1943 theories) to explain the mechanism of production of the clinical and laboratory findings on the basis of adrenal cortical hyperactivity. Albright emphasized three factors which he thought must be present in this syndrome: (1) diabetes (decreased sugar tolerance) resistant to insulin; (2) muscular weakness (with low creatinine excretion as an index of decreased muscle mass), and (3) osteoporosis, especially of the spine, with normal serum phosphatase, hypercalciuria early in the disease, and often nephrolithiasis. The other manifestations, he wrote, are of secondary importance and not always present. His deductions were that hyperplasia of the adrenal cortex should be present in every case of Cushing's syndrome. If not demonstrated he would either doubt the diagnosis or doubt the histopathological report.

Kepler[39] agreed with Albright that the essential feature of the syndrome is hyperadrenocorticism. He admitted, however, that the basophil adenomas may be responsible for the initiation of the adrenal overactivity. He felt also that in those cases where no tumor or other abnormality of any endocrine gland could be demonstrated [12 such cases reported up to 1940: see Cluxton *et al.*[10]] there might still be

hyperactivity of the adrenals without histopathological evidence to confirm it. This constitutes the most acceptable view at present on the subject of the pathological physiology of Cushing's syndrome.

Many other theories as to the causation of Cushing's syndrome have been elaborated, but most of them revolve around either hyperfunction or dysfunction of either the pituitary gland or the adrenal cortex. The unusual bodily configuration (truncal obesity) has been explained as due to kyphosis and shortening of the spine due to the osteoporosis (Freyberg and Newburgh[27]) and in part due to weakness of the abdominal muscles. These interesting suggestions fail to explain the truncal obesity in the patients without osteoporosis, kyphosis or muscular weakness.

Heinbecker[35] reported 5 autopsied cases of Cushing's syndrome, all 5 showing Crooke's changes in the pituitary basophil cells. In 4 he found degeneration of paraventricular hypothalamic nuclei. He reproduced changes similar to Cushing's syndrome in animals by injuries to the hypothalamic nuclei. His theory was that hyalinization of the basophil cells was the intermediate stage in Cushing's syndrome, and could be caused by adrenal cortical tumor, thymic tumor or atrophy of the hypothalamic nuclei, and that Cushing's syndrome was really one of *hypo*pituitarism, explaining the basophil adenomas as attempts at compensation for loss of basophil cells. A more reasonable explanation of his findings would be that through hypothalamic injuries the sympathetic system was activated, exciting the adrenal *medulla* to overactivity, which in turn provoked the adrenocorticotropic hormone production from the pituitary basophil cells and finally hyperactivity and then hyperplasia of the adrenal *cortex*, as outlined on page 9. Whether exhaustion of the basophil cells and subsequent Crooke's changes in them would lead to compensatory basophil hyperplasia and adenoma formation is a moot question, but none the less interesting and stimulating.

Whatever the cause may be, and it is safe to say that at the present time no single cause has been found, the syndrome is still a distinct and definite one, even though there are variations in some clinical features, discrepancies in the laboratory and autopsy findings, and no universal satisfactory method of treatment.

PATHOLOGIC CHANGES

Autopsy findings in 98 cases reviewed by Thompson and Eisenhardt[63] showed:

60 pituitary tumors
22 adrenal tumors
12 no endocrine tumor
3 thymic tumors
1 arrhenoblastoma

As noted above, the authors reviewed the slides on 65 pituitary sections. Nearly all of the pituitary tumors were of basophil cells, and all but 5 of the pituitary glands revealed Crooke's changes. Of the 22 adrenal tumors, 20 were in females. Eighteen of the 22 were operated on and all died in the postoperative period. At that time (before 1940) there was no satisfactory treatment for adrenal insufficiency. Such is now available and fairly well understood.[64,65] It is worth stressing that these patients with Cushing's syndrome stand operation poorly, and are apt to die of uncontrollable infection after operation or injury. In the patients with large hyperfunctioning adrenal tumors, the opposite adrenal is likely to be atrophied or absent, so that removal of the tumor results in acute adrenal insufficiency[59]. The causes of death in other patients have been intracranial hemorrhage, acute pulmonary edema, or pneumonia, and heart failure, often due to the hypertension. Other autopsy findings commonly present are: normal or enlarged thyroid glands; normal or atrophied parathyroid glands (with 2 exceptions); normal or infarcted or hyperplastic adrenals (rarely atrophied); normal or inactive gonads; atrophied or absent thymus (with a few striking exceptions); fatty infiltration of the liver, parathyroids and pancreas; generalized arteriosclerosis;

cardiac enlargement; chronic glomerulonephritis or progressive vascular renal disease, sometimes with small calculi; osteoporosis with normal bone marrow and delayed epiphyseal union in the cases involving children.

LABORATORY FINDINGS

The laboratory findings have not been constant or always consistent, but the majority show:

A. Urine:

1. Glycosuria (or decreased sugar tolerance with normal fasting blood sugar).
2. Albuminuria and casts in some patients.
3. Occasional hypercalciuria, often early in the disease.

B. Blood:

1. Normal or slight to moderate elevation of red blood cell count and hemoglobin.
2. Polymorphonuclear leukocytosis with lymphopenia* and eosinopenia.
3. Phosphorus low normal; calcium high normal.
4. Hematocrits and sedimentation rates normal (in absence of infection).
5. Phosphatase normal (late increase with improvement).
6. Electrolytes normal in some cases. Chloride often shows a fall with an increase in the carbon dioxide combining power and the presence of a moderate degree of alkalosis.
7. Cholesterol normal or elevated.

C. Hormonal assays:

1. Mild to moderate increases in 17-ketosteroid excretion. (This is diagnostically significant in comparison with the excessive excretion of these androgenic breakdown products and of androgens in the adrenogenital syndrome.)
2. Greatly increased excretion of corticosteroids. (This is diagnostic of adrenal cortical overactivity and in conjunction with some of the signs and symptoms of Cushing's syndrome it is diagnostic of it. The diagnosis cannot be made in the presence of a normal urinary corticosteroid excretion.)
3. Estrogens normal.
4. Pregnandiol found in some cases.
5. Various androgens are often somewhat increased but do not reach the levels found in the adrenogenital syndrome.
6. Gonadotropins increased in some (early?) and low in others (late?).
7. The truly diagnostic assay of adrenocorticotropic hormone has, as yet, not been satisfactorily carried out in the clinic.

D. Metabolism:

1. Basal metabolism rates normal or low in some, elevated in others.
2. Nitrogen excretion increased (negative nitrogen balance) in a few, normal in most.
3. Normal calcium excretion in most cases, increased in a few (early?).
4. Loss of calcium and phosphorus from gastrointestinal tract, not improved by giving Vitamin D.
5. Good utilization and retention of intravenous calcium.
6. Urinary creatinine excretion low.

ROENTGEN FINDINGS

The roentgen rays are important in the diagnosis of the syndrome as well as in therapy (see below). Diagnostic roentgen examinations should include the skull, the spine and pelvis, chest and urinary tract. The majority, but by no means all, of the patients have a true osteoporosis as defined by Albright.† This is either limited to or more marked in the skull, spine, pelvis and ribs and may spare the extremities.

* Modern studies, by de la Balze *et al.*[20] have shown that the adrenocortical secretions are antagonistic to lymphatic tissue elsewhere in the body. Thus in Addison's disease we find a relative lymphocytosis, and excess lymphatic tissue in the body. With the opposite state of hyperadrenocorticism the usual finding is lymphopenia, and decreased lymphatic tissues. The 3 cases reported as associated with thymic tumors and Freyberg's case of atrophied adrenals and hypertrophied thymus[25] could be explained by complete exhaustion of the adrenal cortex as noted above.

† Albright defines "osteoporosis" as a condition due to the inability of the organism to form and lay down the protein matrix of bone, quite different from osteomalacia (inability to deposit calcium in the matrix) and osteitis fibrosa cystica (excessive loss of calcium from the bone). By this definition, borne out by many experiments, the osteoporosis of Cushing's disease is therefore not due to excessive activity of the parathyroid glands, as Cushing at first believed.

There may be spontaneous fractures, particularly of the ribs and the vertebral bodies. Some patients have dense endplates on the vertebral bodies. Mild degrees of osteoporosis may escape detection, as there is no accurate measure of calcium content of bone by roentgen methods. A dorsal kyphosis is often the result of collapsed vertebrae. In young patients there is usually delayed epiphyseal growth.

The skull may show the fine diffuse granular mottling of osteoporosis but no localized changes and no evidence of increased intracranial pressure. The sella turcica should be normal in size and shape. (2 exceptions (Fuller and Russell[30])). The lamina dura is intact around the teeth, in contrast to the osteitis fibrosa cystica of hyperparathyroidism, where it is absent.

The ribs frequently are fractured, often with excess callus around them, even though the history of definite injury may be absent. We have been unable to confirm Sussman and Copleman's[60] findings of broadening of the anterior ends of the ribs on roentgen examination with excessive callus found in this area at autopsy. (All 4 of their patients with this finding suffered from adrenal carcinoma.) The lungs and mediastinum should be studied roentgenologically in every case to rule out a tumor or metastasis. The heart will be found to be moderately enlarged in most cases, particularly those of long duration and those with hypertension.

Every patient should have roentgen examination of the genitourinary tract as part of the original investigation, and this should include films of the entire abdomen, as well as intravenous or retrograde pyelograms. A definite tumor mass may be demonstrable as in our Case IV, or the roentgenograms may reveal displacement of one kidney or elevation of one diaphragm, or both together. Perirenal air insufflation is useful in practiced hands, but the diagnostic findings in general are hardly worth the time and trouble required. Exploration of both adrenal areas has been done in many patients in preference to Carelli's procedure. Not infrequently the roentgenograms will reveal small renal calculi, a finding which has not been stressed in the literature.

The frequency of gallstones in patients with this syndrome is unknown, as it is rarely mentioned in the protocols, but we would expect it to be high. Our Case II has gallstones at the age of thirty, and gallstones were found at autopsy in Case III.

CASE REPORTS

Case I. Earl F. (previously reported, Case II in reference 15).

A dentist, aged thirty, was admitted to the Peter Bent Brigham Hospital August 11, 1930, complaining of painful obesity, loss of strength, irritability, polyuria and polyphagia. Family and past history were unessential, patient being married with two children. Present illness began five years before admission, when he noticed he was growing round shouldered and stout, having gained 60 pounds. He dieted and lost weight but became easily fatigued, unable to concentrate, irritable, then depressed. Fat deposits appeared symmetrically on the head, neck and face; then the skin of the face became tense and cyanotic. Eight months before admission excessive thirst, weakness and impotence developed. Seven months before admission glycosuria was discovered.

Physical examination showed a round-shouldered man, appearing older than his stated age. The abdomen was paunchy, the face, neck and trunk were obese, but the extremities by contrast appeared thin. The face was florid and dusky in hue, with tender fatty deposits covered with glistening red skin (Fig. 7). There were numerous purplish striae in the groins and over the abdomen and thighs. The skin over the extremities was pigmented and scaly. Labortory findings revealed a red blood cell count of 4,800,000; white blood cell count of 16,700; hemoglobin 90 to 100 per cent; metabolic rate minus 10 per cent; blood pressure 170 systolic. The urine contained sugar two plus and excess nitrogen, termed "azoturia" by Woodyatt who had studied the patient thoroughly. Blood lipids were increased. Roentgenograms revealed a sella turcica normal in size and shape. No osteoporosis could be demonstrated in the skull, spine or pelvis.

His course, while undergoing elaborate investigation, was steadily retrogressive and he

soon became bedfast, with marked loss of strength, increased pain in the face and hips. A carbuncle developed over the sacrum and did not respond to therapy. At this point, October 14, 1930, a series of four daily roentgen treatments was given to the pituitary region, totalling about 800 r to each temporal area (measured in air). Immediate improvement followed and he was discharged two days after the irradiation was completed. He continued to improve gradually, and was eventually able to return to work. His blood pressure fell to 134/86, the weight decreased, and the glycosuria, polydipsia and polyphagia disappeared.

In spite of subsequent irradiation (1) in February, 1934, at the Presbyterian Hospital in Chicago, Illinois, with eight treatments to each temporal region over a period of six months totalling 2,100 r, (2) September, 1934, at Saint Anthony's Hospital in Rockford, Illinois, with 400 r to each temporal region, and (3) December, 1934, at the New Haven Hospital, New Haven, Connecticut, with 400 r to each temporal region, mild symptoms continued. However, he was able to work until he died suddenly on February 21, 1935, from a spontaneous intrapontine hemorrhage. The body was exhumed after burial at the request of Dr. Cushing, and a postmortem examination was made on March 6, 1935. The pituitary body weighed 1.28 gm. Microscopical examination showed a large basophilic adenoma and the basophilic cells in the anterior lobe showed Crooke's changes. The postmortem showed a slight degree of arteriosclerosis, some skeletal decalcification, and grossly normal appearing adrenal glands.

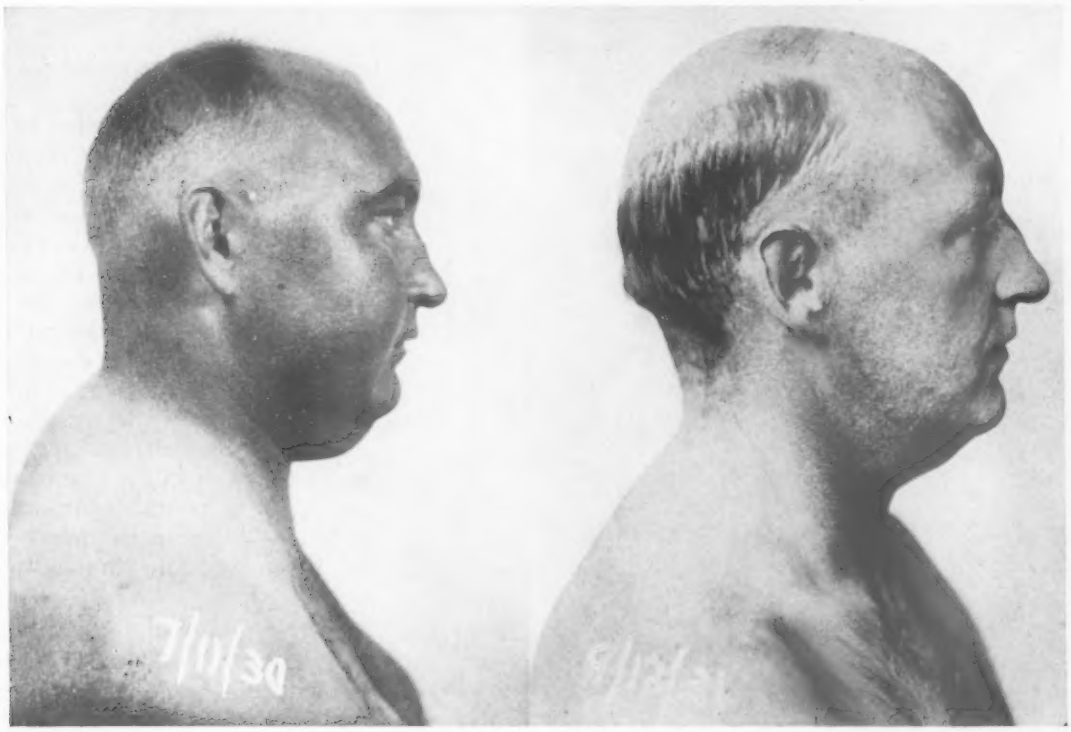

Fig. 7. Case 1, Earl F. before roentgen therapy (July 11, 1930) and after roentgen therapy (September 12, 1931).

Discussion. The immediate response following roentgen therapy of only a moderate amount to the pituitary region was striking and dramatic. Whether it was due to the irradiation or not is another question. Also whether a more prolonged or permanent improvement would have followed repeated or more intensive irradiation as recommended by Freyberg *et al.*[25] is open to question. All of us are apt to fall into the error (probably a form of wishful

thinking) of reasoning that if a certain dose of irradiation gives a certain percentage of good results, an increased dose should give increasingly better results. Unfortunately this is not true, but further experience may tell us whether there is an optimum dose for this and other endocrine tumors, or whether there is a maximum dose, beyond which we do harm rather than good. Certainly this patient did not receive nearly as much irradiation, nor as persistent treatment at first as did the next patient who had a much better result.

Case II. Alice D. (previously reported as Case 14, reference 16).

A fifteen year old girl was admitted to the Peter Bent Brigham Hospital on March 29, 1932, with the complaint of an abscess near the rectum, which was found to be an infected pilonidal sinus.

Fig. 8. Case II, Alice D., before roentgen therapy, April, 1932.

The family history was unimportant. Her past history revealed an appendectomy at the age of eight, followed by an increase in appetite and gain in weight. Menses began at ten years but ceased abruptly in April, 1930, at thirteen years of age. Her weight had increased as follows: twelve years old, 103 pounds; thirteen years old, 122 pounds; fourteen years old, 170 pounds.

Her immediate illness began with a convulsion followed by unconsciousness in December, 1931, followed by a second and similar attack February 29, 1932. After the second convulsion, sugar was found in her urine by Dr. Arthur Cushing of Brookline, who referred her to this hospital.

Physical examination showed a cooperative but lethargic girl, complaining of headaches and backache. Her weight had been reduced by diet to 132 pounds. She was round-shouldered, with adiposity of the face, neck and trunk, but not of the extremities. Her face was dusky red and plethoric, with coarse dark hair on her upper lip, cheeks and chin (Fig. 8). Her skin was dry, dusky and scaly, with cyanosis of hands and feet. There were purple striae distensae over the abdomen, shoulders and thighs, and in the groins. The clitoris was not enlarged.

Laboratory examinations revealed a blood pressure of 140/110, basal metabolic rate of minus 22 to minus 33 per cent, red blood cell count 4,880,000, white blood cell count 9,400, with 87 per cent polymorphonuclears and 13 per cent lymphocytes; hemoglobin 80 per cent (S), a low sugar tolerance with alimentary glycosuria, total proteins 5.4 gm., blood cholesterol 173 mg., nonprotein nitrogen 30.7 mg. per cent, blood calcium 12.1 to 10.0 mg. per 100 cc. Special studies by Dr. Aub showed her to be in nitrogen balance, but in negative calcium balance with an excessive urinary excretion of calcium.

Roentgenographic examinations showed a diffuse osteoporosis, especially of the skull and spine, with compression fractures of several vertebrae. The sella turcica was normal in size and shape. The heart was enlarged. Roentgen treatments were given in eight series of four treatments each, directed through the temporal portals at the sella, as follows:

April	21–25, 1932	4 treatments about 700 r×2
June	2– 6, 1932	4 treatments about 700 r×2
Aug.	1– 4, 1932	4 treatments about 750 r×2
Nov.	1– 4, 1932	4 treatments about 750 r×2
Mar.	1– 4, 1933	4 treatments about 750 r×2
Nov.	6– 9, 1933	4 treatments about 750 r×2
Jan.	10–13, 1934	4 treatments about 750 r×2
Mar.	13–16, 1934	4 treatments about 750 r×2

There was slow and gradual improvement,

although the patient continued to gain weight to August, 1932. The first improvement noted was a change in disposition to a more alert and interested state, and a decrease in backache. Next, the abnormal hair on face, lips and chin began to diminish noticeably by August, 1932. By September her blood pressure was appreciably lowered; in October it was noted that the striae had lost their purple color and in February, 1933, menstruation returned and has been normal and regular since.

The patient has reported regularly for observation and in 1942, ten years after we first made the diagnosis, she was admitted to the hospital for a series of metabolic tests and measurements, all of which were within normal limits. She graduated from high school and started work as a secretary in 1936, was married in 1944, became pregnant in February, 1947, and on November 22, 1947, was delivered of a 5 pound, 14 ounce baby girl by Caesarian section (Fig. 9).

Complete studies in 1947 revealed her to be normal in all respects with three exceptions: (*a*) there still remained slight upper dorsal kyphosis, probably a relic of the previous osteoporosis (Fig. 10), (*b*) numerous small gallstones were discovered by roentgen examination, but are causing no recognizable symptoms, and (*c*) definite skin atrophy, depigmentation, scarring and telangiectases were present in both temporal areas where the irradiation was given.

Discussion. We still cannot call this a "cured" case of pituitary basophilism, but the patient has certainly ceased having the signs and symptoms of the disease. Her improvement followed irradiation of the pituitary region only, with no other form of therapy being given. The amount of irradiation may have been excessive, and she now has skin atrophy and telangiectases in both temporal regions; but if the improvement was due to the irradiation the local damage is but a small price to pay. It might be noted here that Case v, M. G., also has beginning radiodermatitis in each temporal area, with pigmentation and small telangiectases, following four series of treatments (*vide infra*). It seems quite probable that these patients with thin, easily bruisable and easily infected skin may have an increased skin sensitivity to irradiation.

Fig. 9. Case II, Alice D., and baby daughter sixteen years later; May, 1948.

Case III. Miss P. (previously reported as Case 14, reference 17).

The patient, aged thirty-three, was admitted to the Peter Bent Brigham Hospital on October 24, 1932, for study and treatment after a thirteen year history of intermittent and variable polyglandular abnormalities. Her immediate difficulty was amenorrhea for fifteen months, and increased hair on her face.

Her past history revealed that her menses began at thirteen years, and were regular until cessation for twenty months at the age of twenty years. Excess hair on her face was first noted at twenty years of age, improving after the menses returned, but recurring at the age of twenty-five, and requiring a daily shave thereafter. She had been treated by several different physicians with endocrine preparations, with definite but temporary improvement. Eleven years ago there was a marked gain in weight, with the appearance of purplish striae in axillary, inguinal, breast and upper thigh regions. There had been a marked change in her dispo-

sition from a friendly and affable person to a sensitive, highstrung and irritable one. Four months before admission, her face and neck became puffy and swollen, and there was the onset of polyphagia, polydipsia, and polyuria. She also complained of headaches. Physical examination revealed a moon-faced young woman, with a fat neck and striae as noted above. Her skin was dry, with purplish-brown pigmentation on the lower legs. There were several ecchymoses, and a history of easy bruisability of the skin (see photographs in reference 17).

Laboratory findings included a blood pressure of 220/170, traces of albumin and sugar in the urine, low sugar tolerance, red blood cell count 4,600,000; hemoglobin 75 per cent; white blood cell count 7,600; basal metabolic rate minus 10 per cent; blood calcium 10.8 mg. per 100 cc.; blood cholesterol, 192; nonprotein nitrogen 47, fasting blood sugar 99 mg. per 100 cc. Metabolic studies by Dr. Aub revealed normal blood calcium and phosphorus, normal phosphatase, normal fecal calcium and phosphorus, but a high nitrogen output (negative nitrogen balance). The Aschheim-Zondek test was negative.

Roentgen examinations showed an indistinct sella turcica but no enlargement or distortion. There was a diffuse osteoporosis of mild degree and several small renal calculi were demonstrated by roentgenograms in each kidney.

Roentgen therapy was given to the pituitary region November 8–11, 1932, about 610 r to each temporal area, and the patient was discharged to her home to report later. She died suddenly at home December 3, 1932, and autopsy, after embalming, revealed a basophilic adenoma 2.5 by 7.0 mm. in size. The adrenals were hyperplastic, the thyroid was small, no thymus could be found, the uterine mucosa was atrophied, the lymphoid tissue generally was atrophic, and there was advanced arteriosclerosis. Small renal calculi were present in the kidneys.

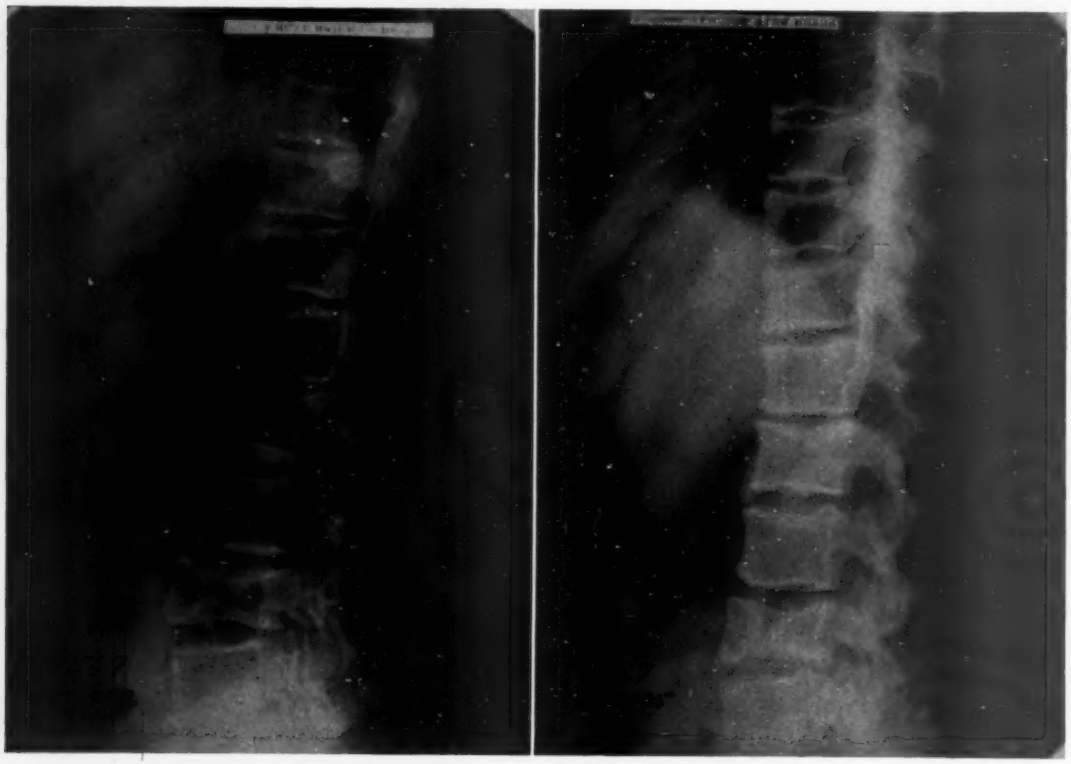

Fig. 10. Case 11. Lumbar spine before (October, 1932) and after (April 1, 1935) roentgen therapy to pituitary area. Note pathological fractures of T12, L1 and L4, also recalcification with clinical improvement.

Discussion. Only three weeks elapsed

between roentgen treatment and death with no evidence of subjective improvement. Case I showed clinical improvement two days after irradiation was completed, but Case II showed no improvement for two months. There was no appreciable histopathological difference between this adenoma and others of the series, which could be ascribed to the irradiation. Sudden death is not uncommon in this syndrome, at times in what seems to be a hypertensive crisis, in others from heart failure and pulmonary edema. There was no really adequate cause found at postmortem for the sudden death.

Case IV. Louise B. (not previously reported. S57086).

This thirty year old married Italian girl was admitted to the Peter Bent Brigham Hospital on March 9, 1938, complaining of sleepiness and blurred vision. Her family history was unimportant.

Her menses began at the age of fourteen, and there was a steady increase in weight from that time. At sixteen years excess hair was noted on the face, and at eighteen years there was increased hairiness on her body. Her weights were as follows: sixteen years, 140 pounds; twenty-one years, 180 pounds; thirty years, 200 pounds. In 1934–1935 there was an eighteen month period of amenorrhea, then two years of regular periods, with amenorrhea again after December, 1937. She had been married nine years with no pregnancy.

Her present acute illness began three weeks before admission with a sore throat, sweating, headache, sleeplessness, easy fatigue and blurred vision. Polydipsia and polyuria were also present. Libido disappeared two weeks before admission.

On physical examination the patient was found to be restless, irritable, and uncooperative. She was 5 feet, 4 inches tall, weighing 200 pounds with a tremendous torso, heavy features, and coarse dark hair on cheeks, chin, lip, chest and abdomen (Fig. 11). There was slight bilateral exophthalmos. Her blood pressure varied from 200/110 to 170/110 but it fell to 140/80 on bed rest. There was slight cyanosis and acne of the face. The clitoris was markedly enlarged. The extremities were well muscled but slender.

Fig. 11. Case IV. Louise B. Hirsutism and obesity in adrenogenital syndrome, not true Cushing's disease.

Laboratory findings included: Red blood cell count 4,620,000, white blood cell count 11,000 to 26,000 (polymorphonuclears 65 to 90 per cent). Two plus sugar was found in the urine at times, negative at other times. The Aschheim-Zondek test was negative. Basal metabolic rate was plus 50 per cent. Hinton test was negative. Blood studies revealed calcium of 8.2 mg. per 100 cc.; phosphorus was 3.8 mg. per 100 cc.; cholesterol 208-131 mg. per 100 cc.; sodium 295 mg. per 100 cc. The androgens excreted in the urine were excessive, varying from 480 to 1,260 international units, compared to a normal of 50 units in twenty-four hours.

Roentgenographic examination revealed a large mass in the left flank, displacing the left kidney downward and the left diaphragm upward—interpreted as a large adrenal tumor. There was no osteoporosis.

Operation was performed March 18, 1938 by Dr. W. C. Quinby. A large tumor of the left adrenal gland weighing 600 grams was found and removed, but the tumor had metastasized, and her course was rapidly retrogressive, with death ensuing on the forty-fourth hospital day.

At autopsy there were metastases to the liver,

lung and retroperitoneal space. The ovaries were normal. The pituitary gland contained a minute chromophobe adenoma, and there were Crooke's changes in the basophil cells. The pars nervosa was invaded by basophil cells.

Bio-assay of portions of the tumor showed an excessive androgen content.

Discussion. This is a classical case of the adrenogenital syndrome, sharing many signs and symptoms and often confused with Cushing's disease. The differential points are obvious: first, the excessive androgen excretion in the twenty-four hour specimen of urine; second, the marked enlargement of the clitoris; third, other signs of virilism, such as bodily hirsuties; and fourth, the lack of muscular weakness. It is interesting to note that Crooke's changes were evident in the pituitary basophil cells.

Case v. Mary G. (previously reported, reference 19).

This fifteen year old Italian girl was admitted to the Peter Bent Brigham Hospital December 26, 1941, for metabolic studies. The chief complaints which brought her to the Out Patient Department were a rapid gain in weight, pain in the back and cessation of menstrual periods for one year. The patient had always been thin and underweight until her menses began at twelve years, when there was an increase in appetite and gain in weight. She had had mild facial acne since the onset of menstruation and the acne became worse during the menstrual period. At thirteen years she noticed increasing hairiness and redness of her face, headaches and backaches, which have persisted since then. There was a gain in weight from 108 pounds in June, 1941, to 148 pounds in December, 1941. Her height was 4 feet 11 inches.

At physical examination she was found to be an obese, shy, disturbed and unhappy girl, with a round plethoric face and numerous spots of acne on face and chin (Fig. 12). There was excess hair, coarse and dark, on cheeks, upper lip and chin. Pinkish striae were found on thighs and buttocks. No masses could be palpated in the obese abdomen. Hands and feet were small. The clitoris was hypertrophied. Visual fields were normal.

Laboratory findings were as follows: Blood pressure 145/80; red blood cell count 6,150,000; hemoglobin 15.2; hematocrit 47 per cent; white blood cell count 10,000 to 21,000 with 90 per cent polymorphonuclears, 9 per cent lymphocytes and 1 per cent eosinophils; smear normal; nonprotein nitrogen 38 mg. per cent, total protein 8.0 gm. (albumin 5.4, globulin 2.6); blood cholesterol 262 to 320 mg. per 100 cc.; blood calcium, phosphorus, chlorides and sodium essentially normal.

Roentgen examination showed a diffuse mottled osteoporosis of skull, spine and pelvis. The sella was normal in size and shape. The lungs were clear, the heart slightly enlarged. Intravenous urograms were normal and there was no evidence of adrenal or pelvic mass.

Therapy. Attempts to influence the disease were made by administration of (*a*) testosterone propionate, (*b*) diethyl stilbestrol, (*c*) roentgen therapy, (*d*) methyl testosterone, (*e*) testosterone—and further roentgen therapy in that order. The results may be summarized as follows: The patient was restored to a positive nitrogen balance during the administration of testosterone propionate and methyl testosterone, but there was no improvement in her general clinical condition. Stilbestrol likewise failed to affect her condition beneficially (see reference 19 for details).

Roentgen therapy was given as follows:

July, 1942 8 treatments 1200 r × 2
Feb., 1943 8 treatments 1040 r × 2
July, 1943 8 treatments 1040 r × 2

Coincident with and subsequent to the first roentgen treatments, signs of improvement occurred. Her ravenous appetite decreased; the acne and hirsutism improved considerably. There was a loss of weight from 199 pounds to 162 pounds, and a decrease in the cyanosis. Her menses returned in March, 1943. In April, 1943, she was again admitted to the metabolic ward and studies were made after oral methyl testosterone and testosterone propionate. There was a worsening of her outward signs of excessive maleness, i.e. increasing acne, hirsutism, cyanosis and gain in weight, with subsequent improvement after cessation of androgenic therapy and the final series of roentgen treatments.

She has continued to improve without further therapy, and at the present time (August 1, 1948) seems to be normal in all respects, seven years after first being seen at this hospital (Fig. 13). She is alert and cooperative; she weighs 144 pounds, and has lost her cyanosis, plethora and acne. She works long hours as a practical nurse,

FIG. 12. Case v, Mary G. Before roentgen therapy (April 16, 1942).

FIG. 13. Case v, Mary G. After roentgen therapy to pituitary region (April 17, 1946).

FIG. 14. Case vi, Walter S. Before roentgen therapy (April 30, 1947).

FIG. 16. Case vii, Loretta D. Before roentgen therapy to pituitary area (January, 1948).

having gone through a strenuous period of training in a small hospital near Boston. The hirsutism is still present and she finds it necessary to shave about three times a week. As noted above, there are areas of brownish pigmentation and beginning telangiectasia in each temporal area where the irradiation was given.

Discussion. This is an excellent clinical result of seven years' duration, in a classical case of Cushing's syndrome. The credit for this therapeutic improvement appears difficult to allot at first sight, as the patient received not only irradiation to the pituitary region but also testosterone and stilbestrol before irradiation, and testosterone subsequently. In spite of the metabolic changes induced by the hormone therapy, there was no improvement in the signs or symptoms of her disease until roentgen therapy was given. After definite improvement had occurred, her condition became worse when testosterone was again administered, then improved and continued to improve after cessation of androgen therapy and the final series of roentgen treatments. We feel that the credit for her improvement should be given to irradiation.

Case VI. Walter S. (not reported previously. Cushing V. A. Hospital No. R2321).*

This unmarried veteran, aged twenty-seven, was referred to the department of Radiology from the Cushing Veterans Administration Hospital with a diagnosis of Cushing's syndrome, for irradiation of the pituitary region. His history, in brief, is as follows: He was admitted on March 3, 1947, to the Cushing Veterans Administration Hospital (Framingham, Mass.) because of a persistent dermatitis of the hands, wrists and right leg, and for evaluation of asymptomatic hypertension. The onset of the former came while he was on Guadalcanal in 1943; it had remained essentially unchanged except for transient periods of improvement. Over the two and one-half year span preceding admission, he had noted a gradual change in body configuration, with enlargement of the abdomen and increasing fullness of the face. These changes were associated with the insidious and successive appearance of generalized weakness, easy bruisability of the skin, decreased perspiration, visual disturbances, skeletal pain, decreased libido, and headache. An elevated blood pressure had been detected on routine physical examination some seventeen months prior to admission. He gave a history of renal colic and the passage of stones from the left and right sides in 1944 and 1945 respectively. Physical examination showed a man 61 inches in height, weighing 124 pounds, with a round red face (Fig. 14) and a small globular body. Temperature, pulse and respirations were normal. Blood pressure was 150/110 to 190/145. The skin on the hands was thin, dry, red and cracked, and lipomatous deposits were apparent in the neck. The thyroid was barely palpable. Visual fields were normal but considerable arteriosclerosis was found in the retinal vessels.

Laboratory findings included the following: red blood cell count 5,205,000; hemoglobin 15.0 Gm.; white blood cell count 9,800 to 14,350, with 58 per cent polymorphonuclears, 41 per cent lymphocytes, and 1 per cent eosinophils; urine contained occasional albumin but no sugar. The glucose tolerance test revealed a normal curve with no glycosuria. Serological findings were negative. Nonprotein nitrogen 40 mg., cholesterol 200 mg. per 100 cc., total protein 8.5 (albumin 4.8, globulin 3.7); phenolsulfonphthalein test 50 per cent in two hours. The glucose-insulin test was abnormal, but on repetition showed a normal flat curve. The Sulkovitch test was two plus, and again two plus after three days of low calcium diet. Blood calcium was 9.5 mg., phosphorus 2.7 mg. per 100 cc.; phosphatase 5.0 Bodansky units. Basal metabolic rate was minus 14, minus 21 per cent. CO_2 was 60 to 78 volumes per cent (26.8–34.8 milliequivalents per liter). Cortin in the urine was 1.8 mg. in twenty-four hours, and repeated on a low protein diet was 1.4 mg. in twenty-four hours.* 17-ketosteroids averaged 9.7 mg. in twenty-four hours; chlorides 90.6 mg.

Roentgenograms of the chest showed old fractures of the third and sixth ribs on the left with callus (Fig. 15). The heart was not en-

* I am indebted to Dr. Maurice B. Strauss and Dr. Carmer Hadley for the details on this patient.

* Tests done by Dr. W. H. Daughaday who wrote: "The value of 1.8 mg. of cortin-like material is slightly elevated by our method (normal 1.0–1.6) but definitely lower than those cases of Cushing's syndrome who did show abnormalities of carbohydrate metabolism. We did not make sufficient observations to determine whether the fall to 1.4 mg. on a low protein diet was significant but it suggests the influence of dietary protein on cortin excretion as suggested by Dr. N. B. Talbot."

Fig. 15. Case VI. Osteoporosis of spine, numerous spontaneous rib fractures, and bilateral renal calculi.

larged and the lungs were clear. The skull showed osteoporosis and a normal sella turcica. The spine was osteoporotic but with no collapse of the vertebrae. A roentgenogram of the abdomen showed a small calculus in the left kidney. Films of the extremities and intravenous urograms were normal.

Put on the rice diet for one week, the patient's blood pressure fell to 110/85, but he complained of headaches and marked weakness and the diet was discontinued. Roentgen treatments of 900 r×2 were given at the Peter Bent Brigham Hospital from April 22 to 30, 1947, with no effect noted in the subsequent seven months. The patient was then readmitted to the Cushing Veterans Administration Hospital with definite evidence of progression of his disease, but still without disturbance of carbohydrate metabolism. Re-examination by roentgen ray revealed spontaneous fracture of the ninth thoracic vertebra.

In November, 1947, the patient was transferred to the Massachusetts General Hospital for perirenal air insufflation and possible exploration of both adrenal areas. Blood and urine chemical and metabolic determinations were essentially unchanged from previous studies. During his preoperative period he suffered a compression fracture of the first lumbar vertebra. Perirenal air insufflation showed a normal-sized right adrenal and an enlarged left adrenal. In December, 1947, approximately 40 per cent of an enlarged but histologically normal left adrenal was excised at operation. The postoperative course was uneventful except for the passage of a small calcium oxalate calculus.

One month later 60 per cent of the right adrenal was excised. The gland appeared normal grossly but microscopically was seen to be hyperplastic. The postoperative period was stormy and characterized by weakness, nausea, vomiting, weight loss, a fall in blood pressure from 125–130/100–110 to 80/50, and the onset of generalized brownish pigmentation accentuated in the palms, antecubital spaces, axillae, and the gluteal folds. Vigorous supportive therapy was necessary during this period, including the use of aqueous adrenal cortical extract, lipoadrenal extract and whole blood transfusions. Four weeks after operation it was noted that his face was less plethoric and that his blood chemistry was normal. Late in his convalescence he passed another ureteral calculus.

In April, 1948, he was readmitted to the Cushing Veterans Administration Hospital where several serum sodium determinations were normal. In August, 1948, his condition was as follows: weight 85½ pounds, height 60 inches, blood pressure 95–105/65–80, pulse varying from 100 to 115. The pigmentation mentioned above was still present. The heart was normal in size and shape as shown roentgenologically. His eosinophil count was still low and his bones still osteoporotic. He was well enough to be home, but was receiving 5 mg. of desoxycorticosterone intramuscularly daily. The Cushing's syndrome seems to have been ameliorated but by no means cured by the partial adrenalectomies.

Discussion. This patient was deliberately given a relatively small dose of roentgen irradiation to his pituitary region (900 r×2) in an attempt to find out if a good clinical result could be obtained at this level of dosage, not repeated. There was neither subjective nor objective evidence of improvement. Instead of giving a second and larger dose of irradiation to the pituitary gland, or a comparable dose to the adrenal areas, he was subjected to partial adrenal-

ectomy, with amelioration of his symptoms as noted above. The result in this patient may be compared with that in the next patient (Case VII) in whom a larger dose was given (1500 r×3). The comparison may be unfair as Case VII is a female, and our best and most lasting improvements have been in females. The underlying disease may be appreciably different in the two sexes in addition to the difference in its outward manifestations.

Case VII. Loretta D. (7A439), aged thirty-seven, mother of two children, entered the Peter Bent Brigham Hospital on December 31, 1947, because of hypertension and diabetes. The patient had been well until her last pregnancy four years before admission when an elevated blood pressure was noted. During the six months prior to admission, she gained 30 pounds in weight, with swelling of the face, neck, trunk, arms and thighs. There was excessive growth of facial hair, and she noted acne of the face and neck, headaches, weakness, and for three months amenorrhea. The physician she consulted found high-grade hypertension and glycosuria and referred her to this hospital for diagnosis and treatment. The past history was noncontributory. There was no family history of hypertension, tumor, diabetes or other endocrine disease.

On examination, the patient was obese, with acne of the face and trunk, excessive facial hair, and purple striae on the abdomen and limbs (Fig. 16). The face had a dusky, plethoric complexion. Blood pressure was 240/130 mm. Hg. There was tenderness of the mid-thoracic spine. The heart and lungs were unremarkable; the abdomen was obese. The visual fields and neurological examination were normal.

The blood Hinton test was negative. The hematocrit was 47 per cent; the white blood cell count 15,000, with 81 per cent neutrophils, 13 per cent lymphocytes and 6 per cent monocytes. The urine contained 1 plus protein and 1 plus sugar. The blood urea nitrogen, total protein, calcium, phosphorus, and alkaline phosphatase were within normal limits. The fasting blood sugar was 197 mg. per 100 cc., the CO_2 combining power 29.9 mm/l, serum chloride 94 milliequivalents per liter, and serum sodium 142.2 milliequivalents per liter. Phenolsulfonphthalein dye excretion was 45 per cent at fifteen minutes and 90 per cent at 120 minutes. Basal metabolic rate was plus 2 per cent. Stools normal.

Roentgenography revealed no abnormality of the skull or chest and no osteoporosis of the spine. Intravenous urograms revealed no evidence of adrenal tumor. The electrocardiogram was normal.

Extensive metabolic studies revealed an abnormally high urinary excretion of 11-oxysteroids, 0.65 mg. in twenty-four hours, and slightly elevated 17-ketosteroids, 22.5 mg. in twenty-four hours. Intravenous glucose tolerance test showed a typical diabetic curve, and the insulin tolerance test demonstrated a relatively insulin resistant diabetes mellitus. Fasting blood eosinophil count was 4 per cu. mm. (normal 100–250 per cu. mm.), indicating excessive adrenocortical function. The adrenocorticotropic hormone test produced a further drop in the partially exhausted reserves of lymphocytes and eosinophils, and the uric acid /creatinine ratio was high and rose only from 0.83 to 1.15.

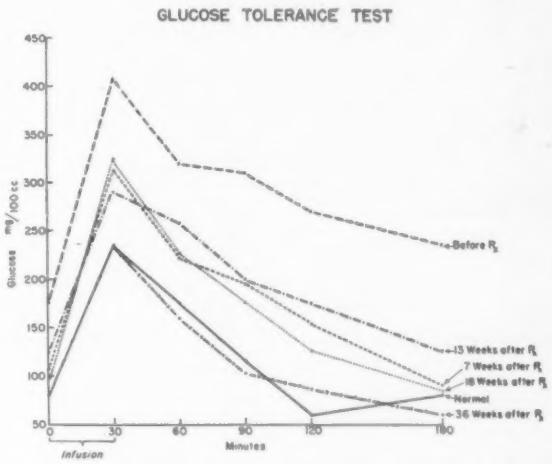

Fig. 17. Case VII. Glucose tolerance curves showing gradual return to normal after irradiation of the pituitary region.

On the basis of these studies, the diagnosis of Cushing's disease was made and the patient was given fifteen daily roentgen treatments, from January 12 to 28, 1948, to the pituitary area, through three portals, for a total dose of 1,500 r×3. Before discharge from the hospital on February 1, 1948, the fasting blood sugar had fallen to 117 mg. per 100 cc. During the subsequent eight months, up to the present writing, the patient has continued to improve with gradual loss of the abnormal facial hair

and striae, diminution of the headaches, and increase in general strength. The glucose tolerance is normal (Fig. 17), and all the metabolic studies have returned to the normal range including a twenty-four hour 17-ketosteroid excretion of 9.8 mg. Menstrual periods have returned and have been regular at three week intervals. The patient's hypertension slowly but gradually improved somewhat. It is of interest that it has been possible to reproduce the values of the pre-irradiation metabolic studies by the repeated administration of adrenocorticotropic hormone for forty-eight hours; that is, a very low eosinophil and lymphocyte count, high uric acid/creatinine ratio and markedly increased 17-ketosteroid excretion up to levels of 53 mg. in twenty-four hours (see Table 1).

TABLE I

SUMMARY OF CHEMICAL CHANGES IN CASE VII AFTER ROENTGEN TREATMENT

(Average values)

4,500 r to pituitary January 12 to 28, 1948

Adm.	Date	Blood Pressure Av. mm. Hg.	Weight	Fasting Blood Sugar mg. per 100 cc.	Eosinophils per cu. mm. (normal 100–250)	Uric Acid / Creatinine Ratio (normal 0.52±0.05)	Leukocyte Count	Polymorphonuclears	Lymphocytes	Hematocrit	CO_2 mm/l	Serum Chloride mEq/l	17-Ketosteroids mg. in 24 hr.	11-Oxysteroids mg. in 24 hr.
1st	1/1	220/130	67.4	202	4	1.10	16,200	80%	15%	47%	29.9	94	23	0.65
After roentgen treatment	1/29	194/112	64.2	105	6	0.83	11,450	—	—	47%	28.7	94	10	—
2nd 7 wk.	3/2 3/7	160/110	69.2 (57.4)	96	47	0.63	10,250	74%	24%	45%	—	—	7	—
3rd 13 wk.	4/12 4/17	174/106	68	100	48	0.56	11,750	73%	24%	46%	28	106	—	—
4th 18 wk. after roentgen treatment	5/24 5/27	168/80	71.2	97	61	0.50	10,800	72%	25%	41%	25	101	6.5	0.14
5th 36 wk.	9/20 9/29	190/110 130/100	68 66.6	70	86 114	0.50	8,600	65%	29 %	44%	24.3	106	9.8	—

THERAPY IN CUSHING'S DISEASE

On the basis of the present day concept of the pathological physiology of Cushing's syndrome two general approaches to therapy readily suggest themselves: (1) the use of measures to counteract those metabolic effects of the pituitary-adrenal cortical axis which lead to pathological changes, and (2) the direct attack on the excessive secretory activity of either the pituitary basophilic cells, the adrenal cortical cells, or both. At present there is no one adequate or satisfactory therapy for Cushing's disease.

In the first approach the prolonged administration of testosterone and its derivatives in an attempt to counterbalance the excessive excretion of corticoids and to reestablish positive nitrogen balance, while the most promising approach, has not proved too successful in the hands of Albright[2], and others.[25,70]

In the second approach the stakes are higher and the results a little more encouraging. The administration of estrogens[25,52,56] represents an attempt at the medical "basophilectomy" in that it is felt that these hormones might suppress the activity of the basophils. Effects are not satisfactory although a few good results have been reported, usually only of a temporary nature. Testosterone might also have a somewhat suppressive action on the basophils and because of its dual action it would appear the agent of choice in what is, by and large, a disappointing therapeutic approach.

In cases which do not show demonstrable tumors of the adrenals, a few good (but sometimes temporary)) results have been reported following the irradiation of the pituitary area or of the adrenal regions. Even in the presence of adrenal tumors one might expect a certain amount of improvement by decreasing pituitary stimulation, but the activity of these tumors is often

independent of any tropic hormones, as any malignant growth would be by definition. The surgical removal of adrenal tumors causing the adrenogenital syndrome has shown good results in many cases, especially in the series at the Mayo Clinic.[68] Partial removal of the adrenals in cases of hyperplasia and Cushing's syndrome, often as a two stage procedure, is fraught with great dangers and is only rarely successful. Adrenal insufficiency often supervenes, especially since a normal adrenal, if present, will show marked involution if the other adrenal is hyperplastic.[59] Such surgical procedures will depend on the availability of adequate adrenal cortical replacement therapy after the operation. Great advances have been made[5,64] and in years to come these procedures might lend themselves to more general use. Meanwhile the therapy of Cushing's syndrome is still an unsatisfactory one. The majority of the patients die of the disease in a few years, and others, after considerable improvement, have relapses. In assaying results of treatment, the tendency of benign endocrine tumors toward spontaneous remissions must always be kept in mind. Cushing's original case, Minnie G. (Case 7, reference 15), was followed from 1910 for many years with minor variations in her condition, and was still alive March 10, 1941.

As soon as the diagnosis of Cushing's syndrome is made or suspected, all reasonable means of proving or excluding an adrenal tumor should be employed, even to bilateral exploration of the adrenal areas, if there is evidence enough to justify it.

The therapeutic attempts in Cushing's disease so far have been irradiation of the pituitary region, irradiation of the adrenal areas, partial adrenalectomy, administration of estrogens or androgens, either singly or together, and occasionally direct surgical attack on the pituitary gland, which is justified in the rare patient in whom roentgenograms show an enlarged sella turcica. Various diets, with and without insulin, such as low sodium—high potassium,[26] low calorie diet,[25] changes in protein, fat and carbohydrate[42] have also been tried. Thyroid and insulin in varying amounts have also been used, but chiefly to treat symptoms of the disease rather than the disease itself. (The diabetes at times is insulin resistant.) Thyroid has not been used in large doses to see if it has any effect on breaking up the abnormal endocrine cycle.

Reports of good results have followed the use of each method but many of the clinical improvements have been only temporary, with a recurrence of symptoms and death as the usual sequel. All methods of therapy fail in the majority of cases, and the disease ends fatally.

It is impossible to sum up the end results in all of the cases reported, except the fatal cases. Many are just case reports over a short period of observation, and temporary improvement is not an adequate reason for recommending any form of therapy. A period of at least five years should be allowed to elapse before evaluating the results as in any disease of prolonged or serious nature.

At the present time, more patients have been benefited, and more prolonged remissions have been obtained, from roentgen irradiation of the pituitary region than from any other method. These good results, however, have been secured in only a small minority of the total number of patients, and the great majority have resisted all forms of therapy. Most of the patients have received roentgen treatments to the pituitary region, many in conjunction with, or before or after other forms of therapy, making it very difficult, if not impossible, to allocate the credit for any benefit which ensues. Complete and lasting remissions are rare indeed, but those obtained by or following roentgen irradiation of the pituitary region seem to outnumber those obtained by any other method. A survey of the available literature reveals that roentgen treatments were given a trial in 42 of the cases reported. Definite improvement followed irradiation rather promptly in 16 patients, or 38 per cent. Seven of these, or 17 per cent, have been prolonged and continued remissions, or, as

Albright so aptly puts it, "have ceased having the signs and symptoms of Cushing's disease."

Finally, in evaluating these and all other results, the peculiar tendency of this disease to cyclic variations and spontaneous remissions[3,63] (as in other endocrine disorders) must always be kept in mind. More patients have had roentgen therapy than any other form of treatment, so that spontaneous remissions, if occurring regularly or in any appreciable percentage, would occur in a higher percentage *coincident* with irradiation of the pituitary body. Even so, it seems rather striking and unusual that of our 6 cases of Cushing's disease,* 4 have had marked improvement following irradiation of the pituitary region. The other 2 cannot be counted as failures of the method as one died a few weeks after treatment, before time enough had elapsed to determine the result, and the other one received only a single series of treatments, with a relatively small dose. Case I, who was markedly improved after roentgen treatment, relapsed and died four years later. Cases II, V and VII have ceased having signs or symptoms of the disease, Case II for sixteen years, Case V for seven years. The exact dose of roentgen radiation which should be given, the quality and the timing are uncertain. Some of the patients reported in the literature have received much more total dosage than our patients, without permanent benefit. Many patients received doses similar to those we gave, also either without effect or with only temporary benefit, and one patient at least, possibly more, received a much smaller dose with a prolonged remission, which

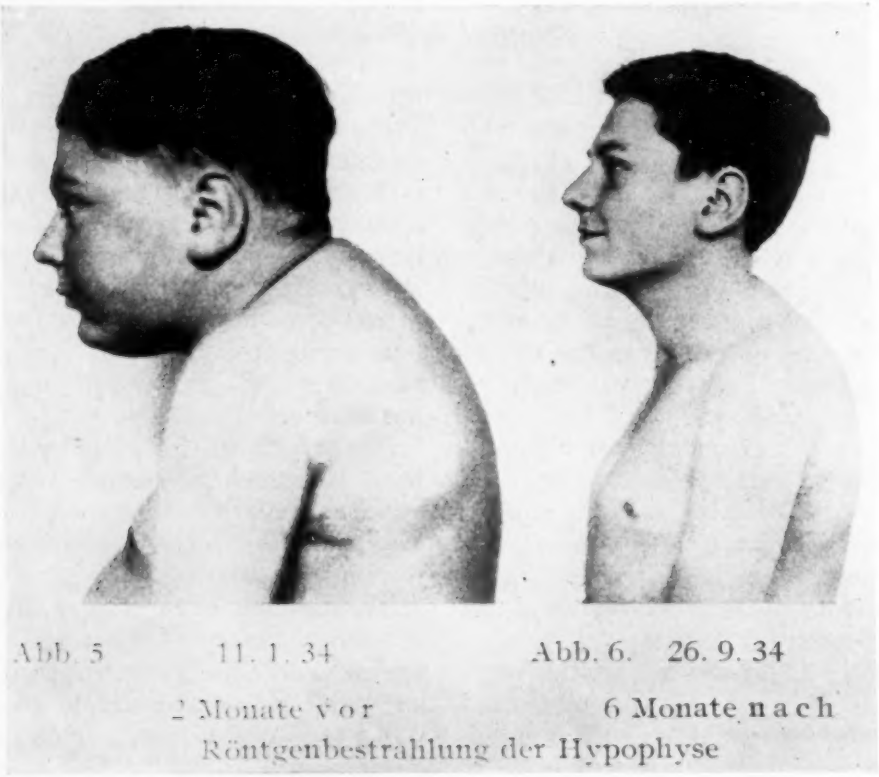

Fig. 18. Jamin's case, two months before and six months after roentgen therapy to the pituitary region.

* Case IV, adrenogenital syndrome due to adrenal tumor, is not considered as a case of Cushing's disease.

TABLE II

BASOPHILISM—CASES IN THE LITERATURE WITH IRRADIATION TO THE PITUITARY GLAND

42 cases reported had roentgen therapy to pituitary region

Age range—at onset of symptoms—8–41 yr., average 23.2 yr.
at death—11–45 yr., average 30.2 yr.

Duration of nonfatal cases when reported: range 1–15 yr., average 4.9 yr.

Sex distribution: 29 females, 12 males.

Clinical features:

Typical appearance	42/42*
Amenorrhea—impotence	38/42
Osteoporosis	34/42
Abnormal sugar metabolism	33/42
Hypertension	40/41
R.B.C. over 6 million	5/28

R.B.C. range 4.3–6.3 million, average 5.2 million

Pathological findings:

Autopsies	19/42
Basophil adenoma	11/20 (one "negative" biopsy)
Crooke's changes	15/15
Adrenal hyperplasia	14/17

Roentgen therapy:

Dose stated	16/42
Two portals	9/13
Three portals	4/13
200–210 kv.	9/14
180–185 kv.	4/14
160 kv.	1/14

Course minimum dose: 380 r×2 (our Case 1)

Course maximum dose: 3800 r via 3 unequal portals (Lawrence)[43]
?6000 r—uncertain if in one course (Cervino)[9]

Doses in the 9 almost cured:

Jamin[38]	450 r×3
Gaertner[33]	500 r×3
Cervino[9]	6000 r total
Our Case II	700 r×2, repeated once; 750 r×2 repeated 6 times
Our Case V	1200 r×2; 1000 r×2; 1040 r×2
Our Case VII	1500 r×3
Luft[46]	3 cases—dose not stated

Results

Total improved	16/42
Improved but relapsed (Our Case 1, Paschkis,[52], Albright[1] Case 1)	3/16
Improvement slight or moderate (Lawrence,[43] Wohl,[71] Whitehead,[69] Wright,[72] Case 2)	4/16
Almost completely cured (Our Cases II, V, VII, Jamin,[38] Gaertner,[33] Cervino,[9] Luft,[46] No. 15, 18, 23)	9/16

might be called a cure, as he died in the German army and autopsy showed no tumor or abnormality of any of the endocrine glands. (The only justification for speaking of a "cure" in this disease is when the patient dies of something else while entirely free of symptoms of Cushing's disease as in Jamin's case[38] (Fig. 18).)

The explanation of the good results of various forms and types of therapy will be difficult if not impossible until the etiology of the disease is determined. The symptoms and signs of the syndrome are those of hyperadrenocorticism, but if the disease began in and was limited to abnormal function of the adrenal glands, there should be no effect from therapy directed to the pituitary gland. The latter has many and diverse functions, with evidence of at least twelve different tropic hormones originating in it, six having been isolated and identified.[23] The adrenotropic hormone could be in excess, stimulating the adrenals, or abnormal in type. Any injury to the pituitary gland could suppress the disordered pituitary function and permit the adrenal functions to return to normal with consequent improvement provided no permanent changes in adrenal structure or function have taken place. Thus Naffziger's patient, reported by Lisser,[45] showed marked improvement for twelve months after removal of portions of the pituitary gland which histopathologically did not show either a basophil adenoma or Crooke's changes. Irradiation to the pituitary gland is another form of damage, sometimes followed by improvement. The action of testosterone or stilbestrol could be explained by suppression of the pituitary gland secretions, as considerable evidence has been built up to show that excessive administration of one hormone reacts on the other endocrine glands. Furthermore, continued thyroid or testosterone injections have been

* The numerator in these stated fractions indicates the number of patients who had the particular symptom or sign, while the denominator indicates the total number in whom the particular information was available. For example, the red blood cell count was given in only 28 cases, and was over 6,000,000 in only 5, hence 5/28.

shown to depress pituitary function with a decrease or disappearance of the basophil cells. The common denominator, or the culprit, in the cases of Cushing's disease could therefore still be the pituitary gland, called the Master Gland of the endocrine series by Cushing, and the balance between the various glands can be either upset at any link in the chain, or attacked at any ring between the links. Thus one can explain the spontaneous remissions, as well as the occasional bizarre result such as the patient reported by Anderson and Haymaker,[3] in whom marked improvement followed simple exploration of the adrenal areas, no tumor being found, and no resection of adrenal tissue being done. The local injury presumably disturbed or upset the abnormal balance between adrenal and pituitary glands. This balance, as Aub[5] states, is not a static or permanent affair, but is constantly changing, and in fact requires changing to meet the demands of changes in environment or other bodily demands. Most of the difficulties in the exact diagnosis of Cushing's disease have now been overcome by the use of (*a*) the accurate measurements of the urinary end products of adrenal excretion, (*b*) the total circulating eosinophil count and the uric acid-creatinine ratio in the urine, and (*c*) the adrenocorticotropic hormone test and the adrenalin test, as reported by Thorn[64,65] and his collaborators. The use and interpretation of these tests are detailed in Case VII, and were also employed in Cases VIII and IX, to be reported later.

"SUSPECT" GROUP

One of the major difficulties in assaying the results of treatment in this syndrome is the proof or certification that the patient really suffers from this condition. There are many patients presenting themselves with one or more classical features of the disease yet who do not have enough of the signs or symptoms to be really included in the group. There is no one feature of the many which characterize Cushing's syndrome which is essential, as we have said before, and there is no one criterion or test which is pathognomonic, unless it is the difficult and complicated assays of the adrenal hormones excreted in the urine.

Three types of patients who are often suspected of having Cushing's syndrome are commonly seen in the larger clinics. They are often assumed to be victims of the syndrome and treated as such. These types, as I have seen them are: (*a*) the dark-skinned, obese, slightly hairy young female, usually resulting from racial or hereditary stock of the same type, and with none of the clinical, roentgenographic or laboratory findings to confirm the tentative diagnosis; (*b*) the red-faced, obese, hypertensive patient, who, if she has amenorrhea or glycosuria or more than her share of down on her face, is often assumed to have Cushing's syndrome without further proof; (*c*) the patient with hypothalamic obesity, often with polyphagia, amenorrhea, abdominal striae and hyperostosis frontalis, even sometimes with glycosuria and hypertension—the most difficult of the three to differentiate from true Cushing's syndrome. The absolute level of circulating eosinophils has proved an excellent diagnostic help, in that patients with active Cushing's disease invariably show a level below 50 eosinophils per cu. mm. (normal 100 to 250), whereas in simple obesity the patient tends to show a normal level.

In addition to the 6 indubitable cases reported above, we have treated 15 others in these "suspect" groups, 7 of them rather closely resembling Cushing's syndrome and the other 8 only remotely akin. It is interesting to note that several of these "basophilism suspects" (as they were classified when the Brigham was a stronghold of the Pituitary Army) have been subjectively improved after roentgen irradiation of the pituitary area. We also treated in 1930–1932 a small series of young patients with essential hypertension by irradiating the pituitary region, using doses similar to those in Cases I and III. No definite or lasting improvement in the hypertension

could be demonstrated, but there were transitory and irregular decreases in the blood pressure, such as may be secured when any form of therapy is first tried.

Those patients were treated by request of Dr. Cushing, who was intrigued by the possibility that eclampsia and essential hypertension might be expressions of basophilism.

DETAILS OF ROENTGEN THERAPY

In Cases I, II, and III roentgen treatments to the pituitary region were given with the following factors:

Voltage 182–185 kv. (peak), mechanical rectifier.
Target-skin distance—40 to 46 cm.
Milliamperes—4 to 6.
Portals—8 cm. circle.
Time—25 to 28 minutes.
Filter—0.5 mm. copper plus 1 mm. aluminum.
Estimated single dose—300 to 350 r.

A "series" of treatments in those early days usually consisted of four daily single treatments, two to each temporal area.

Cases V and VI were irradiated with:

200 kv. (constant potential).
50 cm. distance.
0.5 copper plus 1 mm. aluminum filter.
18 milliamperes.
5 cm. circular portals.
250 to 300 r to 1 portal per day alternating right and left temporal areas. Total dose to each temporal area as indicated, 900 to 1,200 r in 6 to 10 days. All doses measured in air without backscatter. Half-value layer = 0.9 mm. of copper.

Case VII received 1,500 r (measured in air without backscatter) through each of three portals, at the rate of 300 r per day, the other factors being the same as above. Case VIII, now receiving treatment, a young man, aged twenty-nine from the West Roxbury Veterans Administration Hospital, will receive 1,500 r measured in air through each of three portals, which will give an estimated dose in the pituitary gland of 1,700 r. This patient has a classical Cushing's disease by symptoms, signs and laboratory findings, including marked osteoporosis, but without glycosuria or abnormal glucose tolerance curves.

SUMMARY

Seven cases of Cushing's syndrome are presented in detail, six of them definite pituitary basophilism (Cushing's disease), the other one proved to be one of adrenogenital syndrome due to a malignant adrenal tumor. Roentgen irradiation of the pituitary gland was followed by definite improvement in four of the six patients so treated. Two of the patients have returned to normal and have remained well for sixteen years and six years respectively (Cases II, and V). One patient, after marked improvement, had a relapse and died five years later (Case I). The fourth patient, who has shown marked improvement, is too recent to be evaluated as an end result (Case VII). The eighth and ninth patients are undergoing roentgen therapy at present.

The amount, the quality and the timing of roentgen irradiation to the pituitary gland has been variable within wide limits. A summary of the cases from the literature shows even wider variation. Only one-third of the reported cases have shown improvement following roentgen irradiation to the pituitary gland.

CONCLUSIONS

1. If a patient is suspected of having Cushing's disease, the diagnosis can now be made with a high degree of certainty by (*a*) urinary hormone assays, (*b*) the uric acid-creatinine ratio, and (*c*) the adrenocorticotropic hormone test and the adrenalin test, in addition to the other clinical and laboratory findings noted above.

2. We believe the syndrome to be a definite clinical entity, but quite variable in individual patients. There is no single feature or combination of features which is essential to diagnosis in all patients.

3. The duration of the disease and its severity are probably important factors in treatment and also prognosis. Initiated primarily by excessive secretion of adreno-

corticotropic hormone by the pituitary gland, the course of the disease can probably be stopped or reversed by prompt roentgen irradiation to the region of the pituitary gland early in the course of the disease. After prolonged stimulation by adrenocorticotropic hormone the adrenal cortices may become hyperplastic so that their hyperfunction becomes independent of pituitary stimulation. The disease then would have to be attacked at the adrenal level.

4. Our plan of treatment at present (August, 1948) is as follows:

(a) Establishment of diagnosis.
(b) Investigation of adrenal areas to rule out adrenal tumor. If this can be done,
(c) Irradiation of pituitary gland.
(d) If no improvement in signs, symptoms or laboratory findings in three months,
(e) Irradiation to or surgical exploration of adrenal glands. If no tumor or marked hyperplasia be found:
(f) Biopsy of adrenal glands followed by roentgen irradiation to both adrenal glands, following the patient's response closely with the three tests noted above.

5. The signs and symptoms of the disease should be ameliorated in at least half of the patients by this program, and prolonged, possibly permanent remissions should be achieved in at least a third of the cases. Permanent or irreversible changes may have taken place in the other patients before therapy is instituted.

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THE DEVELOPMENT OF THE TAUTOGRAPH AND THE ADVANTAGES OF AUTOMATIZATION IN CARDIOVASCULAR ANGIOGRAPHY*

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HERETOFORE, the success of angiography has depended on estimating accurately the time at which the diodrast would reach a particular chamber of the heart or great vessel and making the roentgen-ray exposure at that precise moment. Physiological tests were used to calculate the circulatory time and, while fairly satisfactory, they consumed the operator's time and usually did not give an indication of what type of congenital shunt or shunts might be present in the circulation.

Angiographic procedures have been further handicapped by the lack of automatic roentgenographic equipment that was capable of making a series of six, eight, ten, or twelve exposures at an interval of one second or less and synchronized with a Potter-Bucky grid. Hence, if angiography was to become a successful routine roentgenographic procedure, the above problems had to be overcome. To do this it was necessary to construct equipment that could take a series of six to twelve roentgen films at one second intervals or less and thus obtain a continuous series of films picturing the bolus of diodrast as it circulated through the chambers of the heart and lungs and into the aorta. In this way the chambers of the heart, the positions or transpositions of the great vessels, the congenital shunts, and the abnormal sequences in the time of visualization of the various chambers of the heart could be recorded on a series of films for study without regard to a preestimated circulation time or the fear of "missing" the diodrast at the crucial moment.

The "tautograph" (Fig. 1) was designed to fulfill these purposes and offers the following advances:

1. It produces a sequence of six to ten angiograms at a one second interval to give a continuous picture of the bolus of diodrast as it circulates through the chambers of the heart and lungs and into the aorta.

2. It eliminates "forecasting" the circulation time and guessing the "route" through the heart.

3. It eliminates estimating the exposure time for only one, two or three films which at best do not provide a complete record of the cardiac circulation.

4. It reduces the number of repeated injections of diodrast that was formerly required to obtain angiograms with the diodrast in different cardiac chambers and great vessels. One injection of diodrast is now sufficient for recording the cardiopulmonary circulation.

5. It eliminates "technician crews" that were necessary for the operation of manual angiographic equipment that could make six to ten exposures.

6. It achieves added clarity and contrast in the films by using a self-cocking Potter-Bucky grid.

7. It has simplified the roentgenographic technique by completely automatizing the advance of the unexposed cassettes, the energizing of the Potter-Bucky grid, the initiating and ending of the roentgen-ray exposure, and the retiring of the exposed cassette.

8. It permits the taking of angiograms with the patient in the semi-erect or in the horizontal position. This is an important

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advantage in the examination of children and infants under anesthesia.

9. It reduces the hazard of excessive roentgen-ray exposure to the patient as roentgenoscopy is not employed.

The "tautograph" was designed by one of us (W.G.S.) and constructed in the shop of Mr. R. H. Tontrup. This equipment consists of a tilting diagnostic roentgenographic table with angle irons fitted under the top to which rollers were fastened for carrying the cassettes. A heavy roller chain with steel lugs spaced at 14 inches carries the cassettes into position and stops the cassette for ⅓ of a second during which a roentgen-ray exposure of 1/15 to 1/20 of a second is made. The cassette is then moved forward and retired into the receiving bin while the next is brought into position for exposure. The time interval consumed in the advance of the cassette, in its stoppage, exposure, and retirement is one second or less. The chain conveyor is energized by an electric motor which operates through reduction gears. The roentgen-ray time exposure is initiated by the self-cocking Potter-Bucky grid, which, in turn, is controlled by a contact switch on the power sprocket connected to the reduction unit.

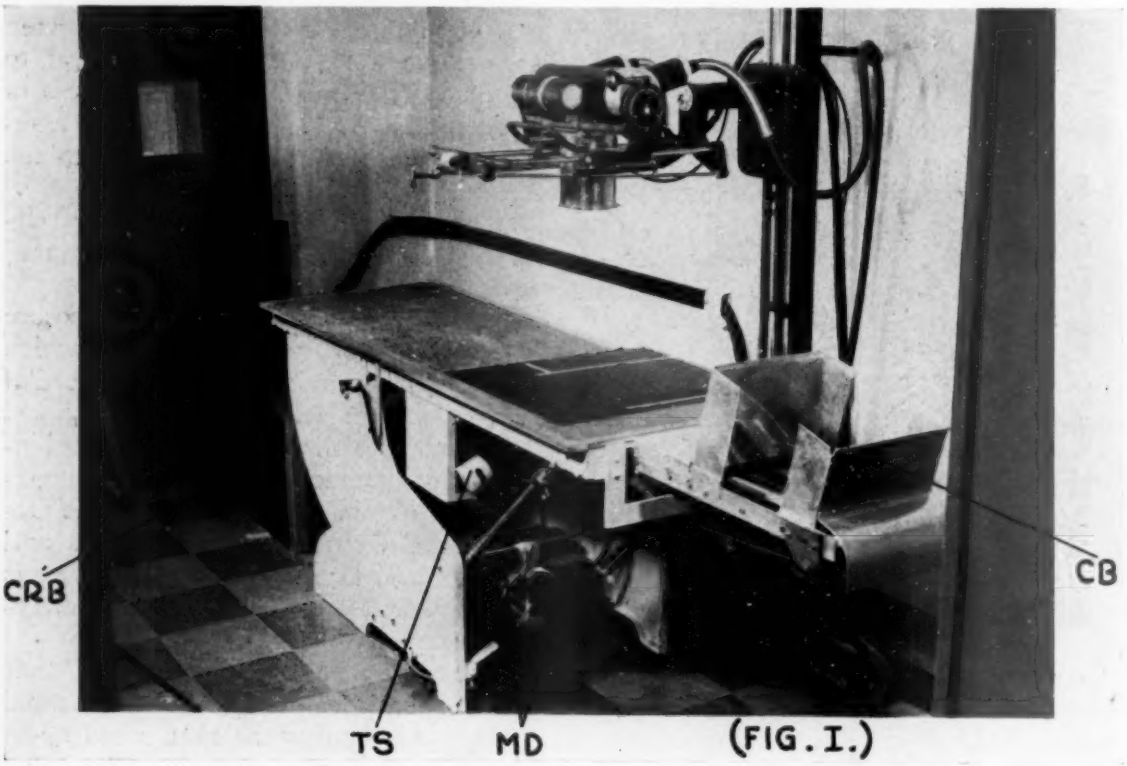

FIG. 1. Side view of the tautograph showing the position of the tube stand. *CB*, cassette loading bin; *CRB*, cassette receiving bin; *TS*, switch for tilting table from horizontal to semi-erect position; *MD*, motor drive for tilting mechanism of table.

With the development of cardiac surgery and the greater necessity for the accurate diagnosis of congenital heart disease an increased need developed for angiocardiography. To meet this demand we realized that special equipment would be necessary to make angiography a practical and successful routine procedure.

Late in 1945 one of us (W.G.S.) had

made preliminary sketches of the tautograph. In July, 1946, we asked Mr. R. H. Tontrup, who had assisted us in the development of the Kieffer-Moore laminagraph, to undertake construction of this equipment. Funds were limited and it was necessary to develop the machine with as little expense as possible. To this end we found a used Kelley-Koett Model "S" tilting diagnostic roentgenographic table and began altering it for our purpose by fitting it with $1\frac{1}{2}$ inch steel angle irons (Fig. 3, 4, 5) running parallel to the sides of the table

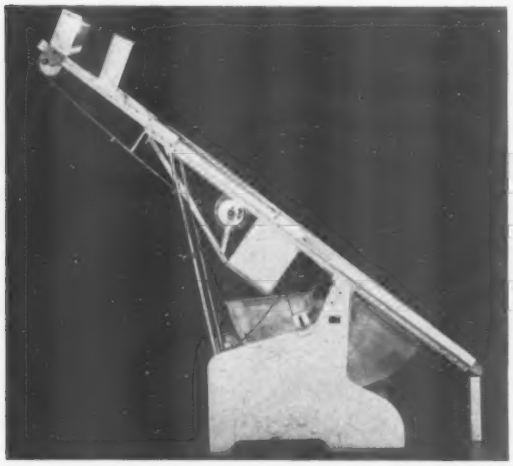

Fig. 2. Side view of tautograph tilted in the semi-erect position at 45°. The table top and the Potter-Bucky grid have been removed.

and spaced the exact width of a Kelley-Koett 11 by 14 inch cassette.

The 11 by 14 inch cassette was selected as the most desirable size since it was sufficiently large to cover the heart and great vessels on an adult as well as on a child and weighed much less than the large 14 by 17 inch cassette. The weight factor was important, for even the 11 by 14 inch cassette weighed $6\frac{1}{2}$ pounds and with ten of them stacked together added a total of 65 pounds to the weight of the table. Furthermore, in our system at least four cassettes would be on the conveyor system at any one moment. One can readily appreciate that considerable force is necessary to move them quickly in rapid succession. Thought was given to devising a special light-weight cassette, but this would have involved additional expense, prolonged the time and

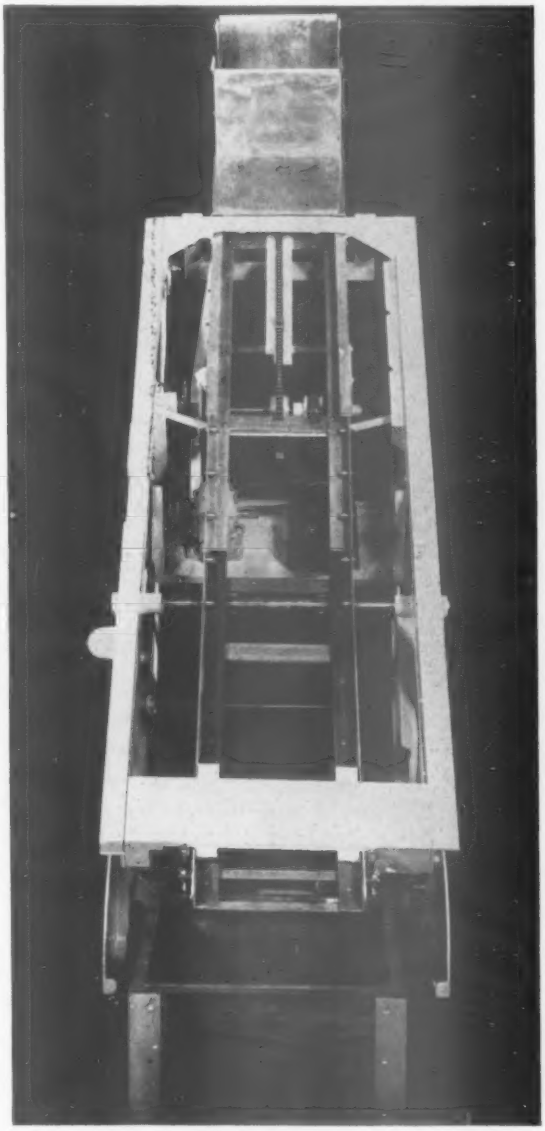

Fig. 3. End view of the tautograph as it is tilted in the 45° position with the table top and the Potter-Bucky grid removed to illustrate the angle irons and brass rollers on which the cassettes are carried and the chain conveyor for moving the cassettes into position.

would have been non-standard items.

To expedite the moving of the cassettes along the channel irons, $\frac{5}{8}$ inch brass rollers (*BR*) (Fig. 4 and 5) were spaced every 5

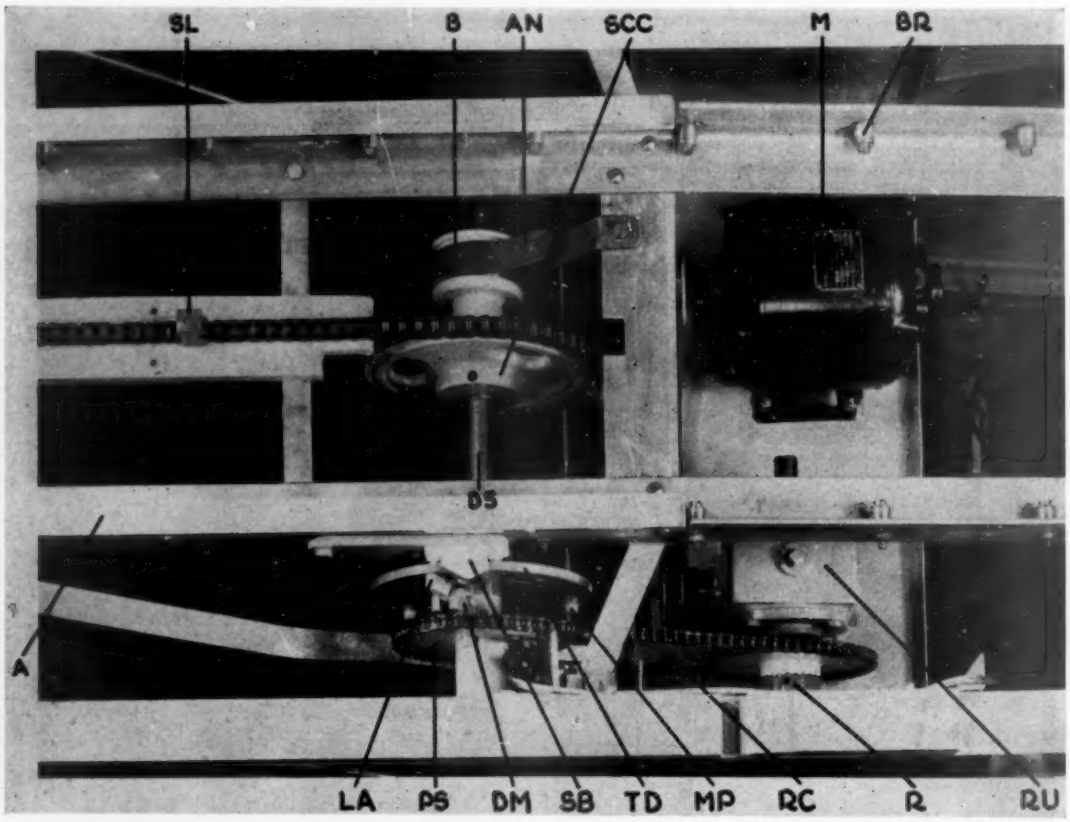

FIG. 4. Detailed vertical view of the conveying mechanism of the tautograph with the table top removed.

SL Steel lug. A cassette is placed between two such steel lugs.
B Brake on drive shaft to provide instant stoppage and elimination of back lash.
AN Adjustable screw for tightening or loosening the brake band.
SCC Steel sprocket which powers the roller chain for conveying the cassettes.
M ¼ h.p. single phase Emerson electric motor.
BR ⅝ inch brass roller.
DS Drive shaft.
A 1½ inch steel angle iron on which brass rollers are secured for carrying the cassettes.
LA Long arm of right angle bar of dog mechanism.
PS Power sprocket.
DM Dog mechanism on power sprocket.
SB Steel block or cam for interrupting transmission of power to the drive shaft.
TD Timing disc.
MP Metal pin in timing disc against which *LA* strikes to revolve the drive shaft.
RC ⅜ inch pitch roller chain extending between reduction unit and power sprocket.
R Ratchet to permit adjustment of cassette conveyor system without turning the reduction unit and motor.
RU Winfield H. Smith speed-reducing unit No. 3½B.

inches along the sides of the channel irons to permit the cassettes to roll along easily.

Knowing the weight and the number of cassettes to be moved and establishing the rate of exposure as one per second, the next problem was to calculate the size of the electric motor that was necessary and to devise some means of slowing down the motor speed for the drive shaft (*DS*) (Fig. 4 and 6) of our conveyor system. After several trials a single phase, ¼ horse power, 60 cycle, 1,725 r.p.m. motor was mounted on a steel cross plate beneath the table and connected by a rubber "V" belt to a

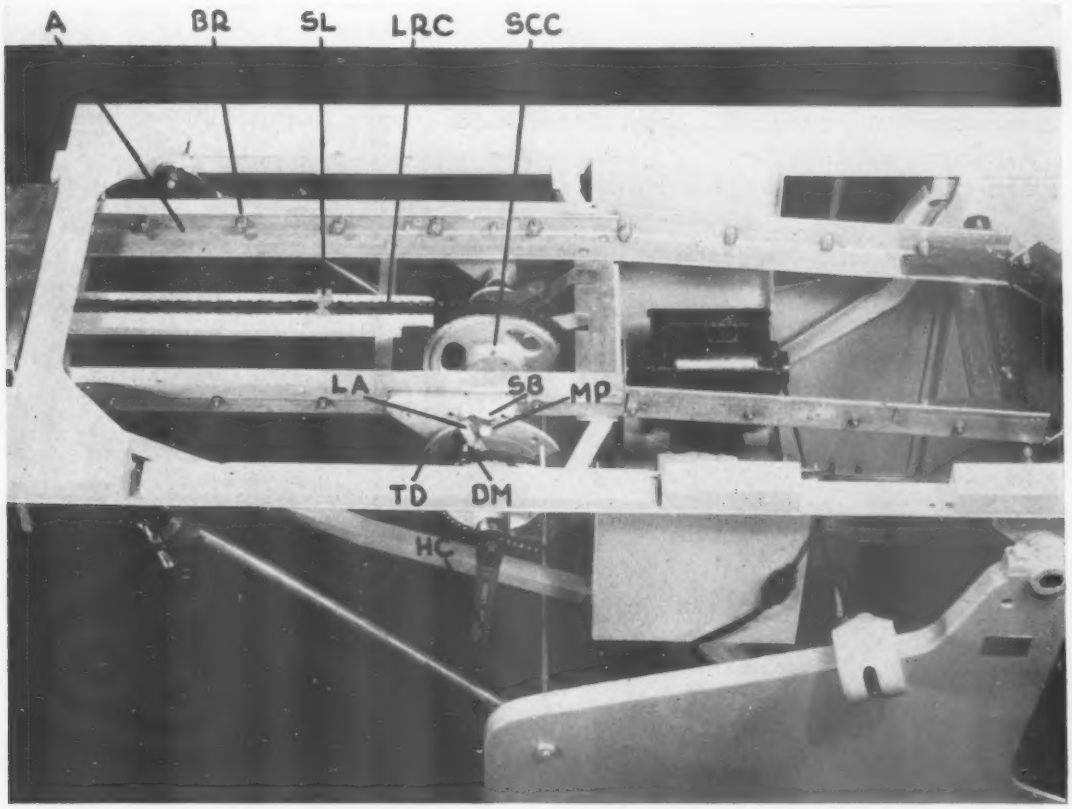

Fig. 5. Detailed side view from above with the table top and bucky removed to illustrate the power and conveying units.

A 1½ inch steel angle iron on which brass rollers are secured for carrying the cassettes.
BR ⅝ inch brass roller.
SL Steel lug. A cassette is placed between two such steel lugs.
LRC ½ inch pitch roller chain for conveying the cassettes.
SCC Steel sprocket which powers the roller chain for conveying the cassettes.
LA Long arm of right angle bar of dog mechanism.
SB Steel block or cam for interrupting transmission of power to the drive shaft.
MP Metal pin in timing disc against which *LA* strikes to revolve the drive shaft.
TD Timing disc.
DM Dog mechanism on power sprocket.
HC Hand crank for adjusting steel lugs to permit proper placing of the first cassette in the loading bin.

Winfield H. Smith speed reducing unit No. 3½B (*RU*) slowing the speed in a ratio of 30:1. With the gear box operating at a constant and known speed, it was possible to calculate the size of the sprocket for the drive shaft of the reduction unit and that for the power sprocket (*PS*) (Fig. 4) of the conveyor system. The two sprockets were connected by a standard ⅜ inch pitch roller roller chain (*RC*).

On the drive shaft of the conveyor system (Fig. 4) the following four devices were placed:

1. The power sprocket (*PS*) that receives the chain from the reduction unit. Note carefully that the axle for this sprocket is a separate housing surrounding and revolving independently of the drive shaft.

2. The timing disc (*TD*) 7½ inches in diameter for starting and stopping the cassette conveying system together with the

trigger mechanism for energizing the self-cocking Potter-Bucky grid.

3. The steel sprocket (*SCC*) $7\frac{1}{2}$ inches in diameter which powers the roller chain that moves and stops the cassettes.

4. The brake (*B*) to induce immediate stoppage of the cassette conveyor system and for eliminating back-lash.

The power sprocket that is chained to the reduction unit revolves on a separate housing surrounding the main drive shaft of the conveyor system. This must be carefully noted as this arrangement is necessary in order to stop the conveyor system for the roentgen-ray exposures.

The movement of the power sprocket (*PS*) is transmitted through a dog mechanism (*DM*), a small right-angle steel bar that is secured to this sprocket by a single metal pin (*PDM*) at the angle of the bar. The long arm (*LA*) of the right-angle bar of the dog mechanism strikes one of three equally spaced metal pins (*MP*) on the timing disc immediately adjacent to it. As long as this arm of the angle bar is in contact with the metal pin of the timing wheel, the main drive shaft is revolving and the cassettes are in motion. With each revolution the long arm of the angle bar strikes a steel block or cam (*SB*) (Fig. 5 and 6)

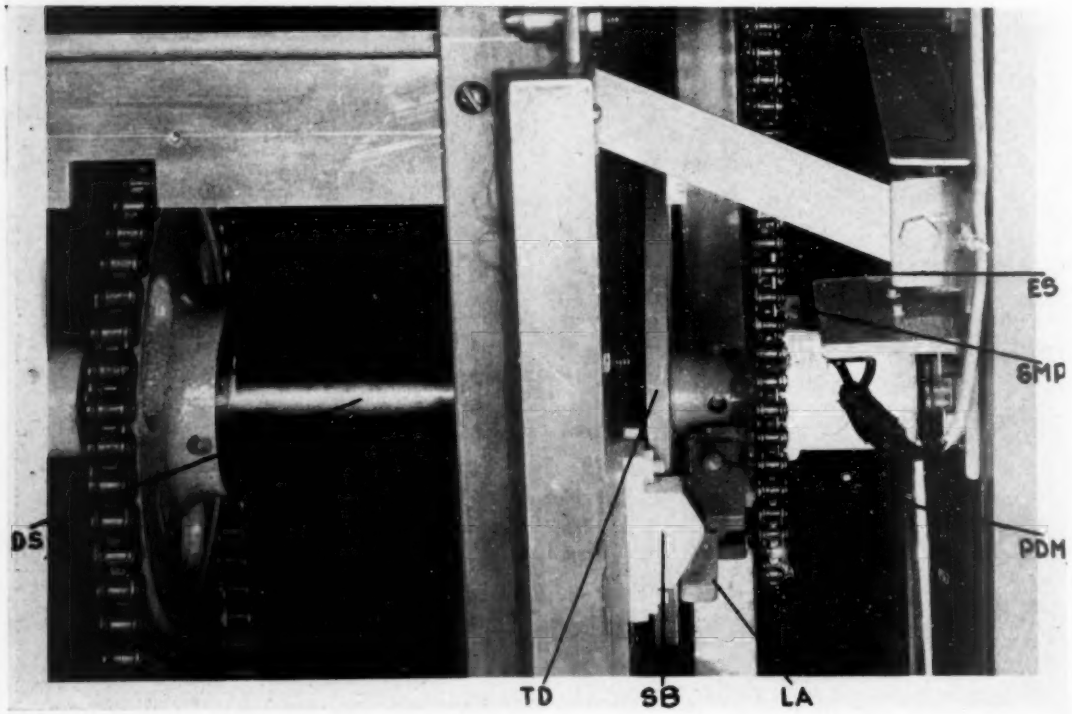

Fig. 6. Detailed vertical views of the timing mechanism.

DS Drive shaft.
TD Timing disc.
SB Steel block or cam for interrupting transmission of power to the drive shaft.
LA Long arm of right angle bar of dog mechanism.
PDM Metal pin placed through the long arm of the dog mechanism.
SMP Small metal pin on power sprocket which strikes electrical switch (*ES*) at the moment that the long arm (*LA*) of the dog mechanism (*DM*) becomes disengaged from the metal pin on the timing disc after it begins to move laterally on striking the steel block or cam (*SB*).
ES Electrical contact which is closed by the metal pin (*SMP*) to energize the Potter-Bucky diaphragm which in turn, by means of conventional electrical contacts starts the roentgen-ray exposure and shuts it off.

fastened to the outside of the long arm of the angle iron (*A*). This steel block is roughly triangular in outline with the base secured to the angle iron and the apex directed toward the power sprocket. As the long arm (*LA*) of the angle iron strikes this steel block (*SB*) it is moved away from the timing disc (*TD*) and is thus disengaged from the metal pin (*MP*) on the timing disc. the steel block again, which stops the conveyor system for the next roentgen-ray exposure.

At the same instant that the angle iron strikes the steel block to stop the conveyor system a small metal pin (*SMP*) (Fig. 6) on the outside of the power sprocket strikes an electrical switch (*ES*) to close the contact which energizes the Potter-Bucky grid

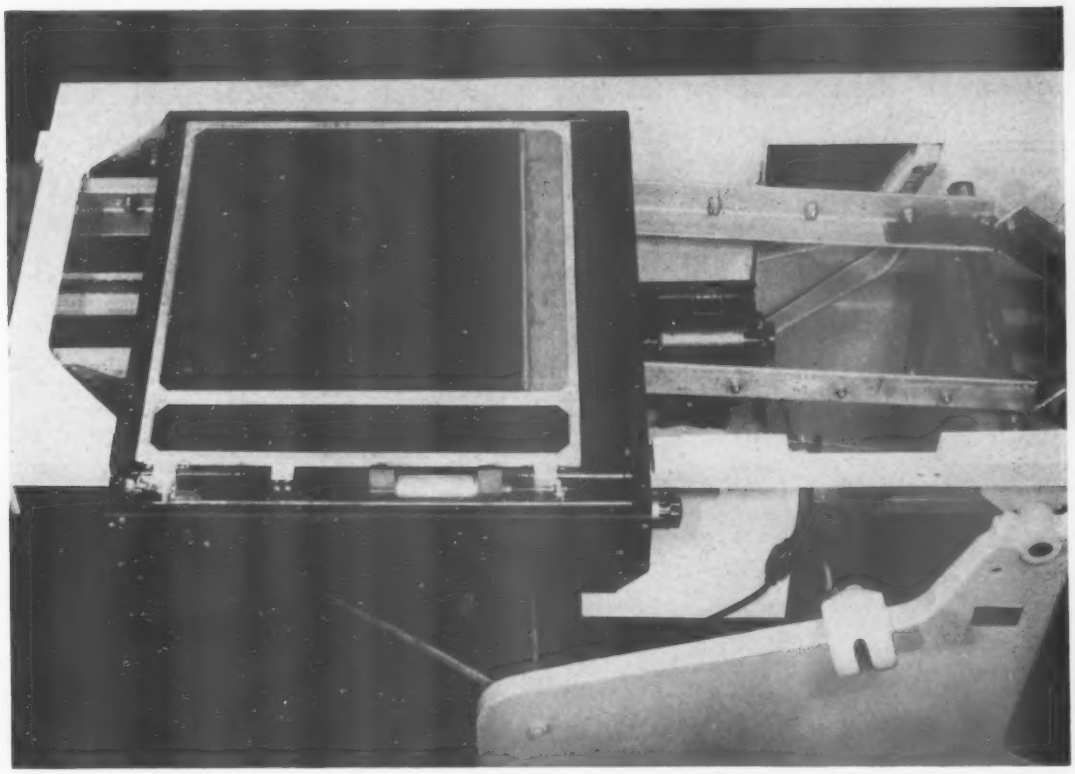

Fig. 7. Detailed view of the Liebel-Flarsheim self-cocking Potter-Bucky diaphragm. The top of the grid and the top of the table have been removed. The grid has been cut down and recentered to handle 11 by 14 inch cassettes instead of the 14 by 17 inch cassettes for which it was originally made.

This action interrupts the transmission of the motion from the power sprocket to the timing disc, and the conveyor system is halted for a brief instant ($\frac{1}{3}$ of a second) until the long arm of the angle iron has passed over the steel block and revolved far enough to strike the next metal pin on the timing disc. On striking the pin, motion is again imparted to the drive shaft and the conveyor system is set in motion until the pin makes a complete revolution and strikes to start it on the down motion. The customary electrical contacts in the Potter-Bucky grid in turn start and shut off the roentgen-ray exposure.

The sprocket (*SCC*) which powers the roller chain for conveying the cassettes is secured to the drive shaft (*DS*). The opposite end of the roller chain revolves around an idling sprocket placed beneath the cassette bin at the extreme end of the table. Steel lugs (*SL*) are placed on the

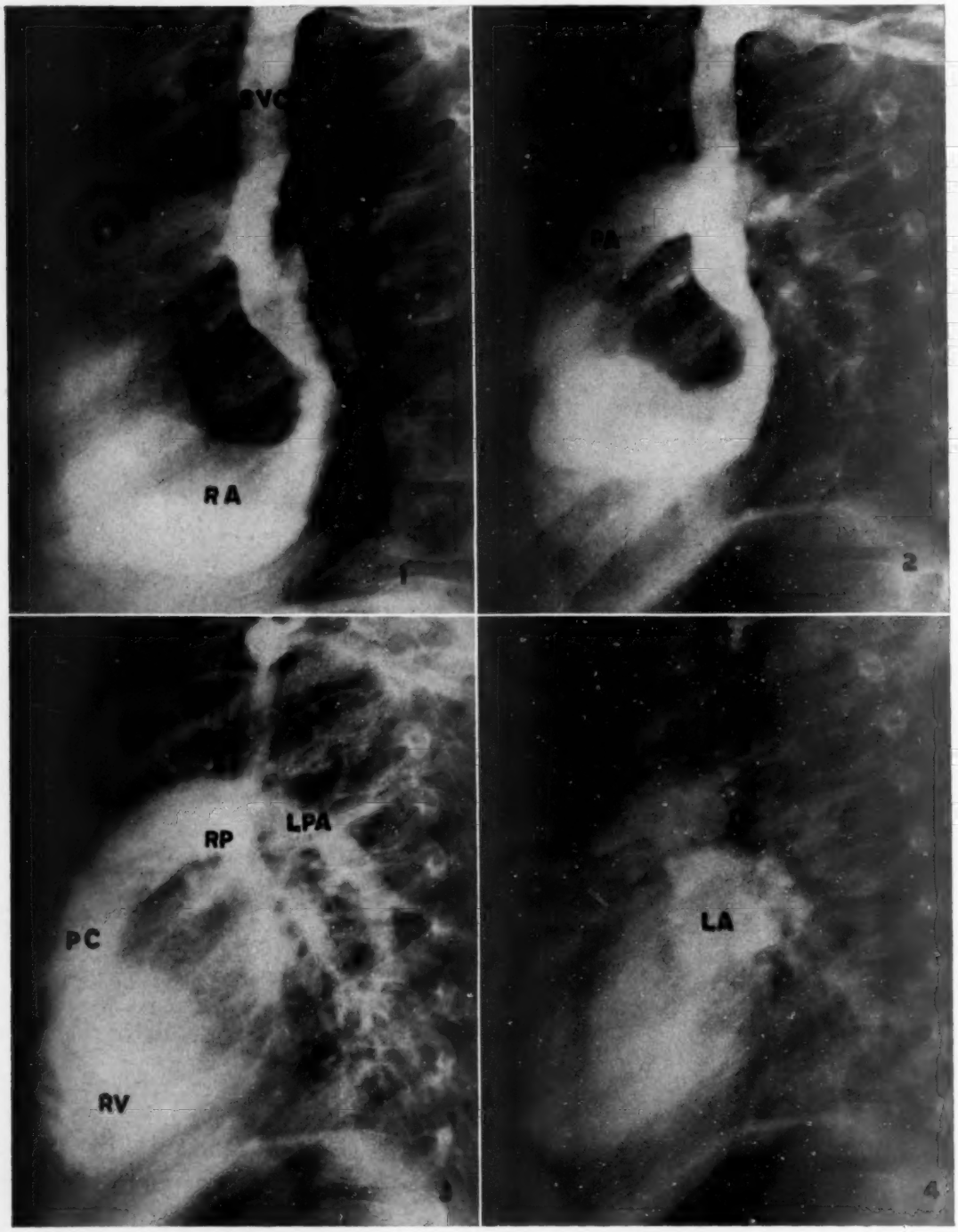

FIG. 8. Normal cardiovascular angiograms taken on the tautograph with the superior vena cava on the left side.

The routine examination includes a series of ten (11 by 14 inch) films made at one second intervals. The tautograph provides a continuous series of films demonstrating the transport of the diodrast through the chambers of the heart and the great vessels. Twenty cubic centimeters of seventy per cent diodrast was injected into the left antecubital vein with the child in the right posterior oblique position and the tauto-

roller chain at just sufficient intervals to permit an 11 by 14 inch cassette to be placed between them. The 14 inch dimension of the cassette parallels the chain.

Since the original photographs were made the brake (*B*) on the drive shaft has been widened to 1½ inches to provide greater efficiency and smoother operation. It is simply a steel band covered with conventional automobile brake lining placed around a flat pulley on the drive shaft. An adjustable nut (*AN*) is available for tightening or reducing the efficiency of the brake as the occasion demands.

A hand crank (*HC*) (Fig. 5) with a ratchet device (*R*) was placed on the outside of the sprocket on the reduction unit so that the conveyor system could be moved by hand. It also serves to move the conveyor chain into the proper position for placing the first cassette between two of the steel lugs. The remaining cassettes are stacked in numbered sequence and drop down on the conveyor chain as soon as the next steel lug moves into place.

The retirement of the cassettes is provided for by tilting the end of the channel irons toward the floor so that the cassettes can slide by gravity to a partially soundproofed receiving bin (*CRB*). The wooden receiving bin is lined with foam rubber to protect the frames of the cassettes.

The self-cocking Potter-Bucky grid is the standard model made by the Liebel-Flarsheim Company except that the grid was cut down and recentered to conform to an 11 by 14 inch cassette. In our experience this grid adds desirable contrast to the films and is worth the additional expense. In all children and infants the roentgen-ray exposure is reduced to 1/20 of a second and in most adults to 1/15 of a second although 1/10 of a second is required for large patients.

A rotating anode tube is used for greater definition. All the power that is available is desirable. We use 500 milliamperes, 65 to 100 kilovolts with exposure times of 1/10, 1/15, or 1/20 second.

Figures 8, 9, and 10 are a series of ten films made on the tautograph at one second intervals. These films, taken in rapid sequence, demonstrate the progress of the opaque chemical through the chambers of the heart and great vessels. The advantage of this procedure is apparent from an examination of the films. The technical quality has been greatly enhanced by incorporating the self-cocking Potter-Bucky grid and synchronizing it with the roentgen-ray exposure. Originally we felt these children had to be anesthetized, but subsequent experience has shown that most of them can be satisfactorily handled by giving a little phenobarbital to the older children a short time before the examina-

graph horizontal. The films were made at a rate of slightly less than one per second and with a self-cocking Potter-Bucky grid synchronized with the exposure.

This case is a boy aged five and a half. He was well developed and never cyanotic. He had a systolic murmur over the precordium and further studies suggested that he might have an aortic or subaortic stenosis, but these possibilities were ruled out by angiography. The progress of the diodrast is well demonstrated and can be followed through the cardiovascular cicrulation by consecutive examination of the films. The following abbreviations designate the different chambers and great vessels.

SVC—Superior vena cava	*PC*—Pulmonary conus	*BA* —Bulbus arteriosus
RA —Right auricle	*LA*—Left auricle	*LPA*—Left pulmonary artery
RV —Right ventricle	*LV*—Left ventricle	*RP* —Right pulmonary artery

Film 1. The diodrast is in the superior vena cava, the right auricle, and right ventricle and is entering the pulmonary conus.

Film 2. The right ventricle and pulmonary artery are better filled.

Film 3. The right ventricle is now completely filled and the contrast medium has progressed into the pulmonary arteries.

Film 4. The diodrast is returning through the pulmonary veins, is filling the left auricle and a small amount has entered the left ventricle.

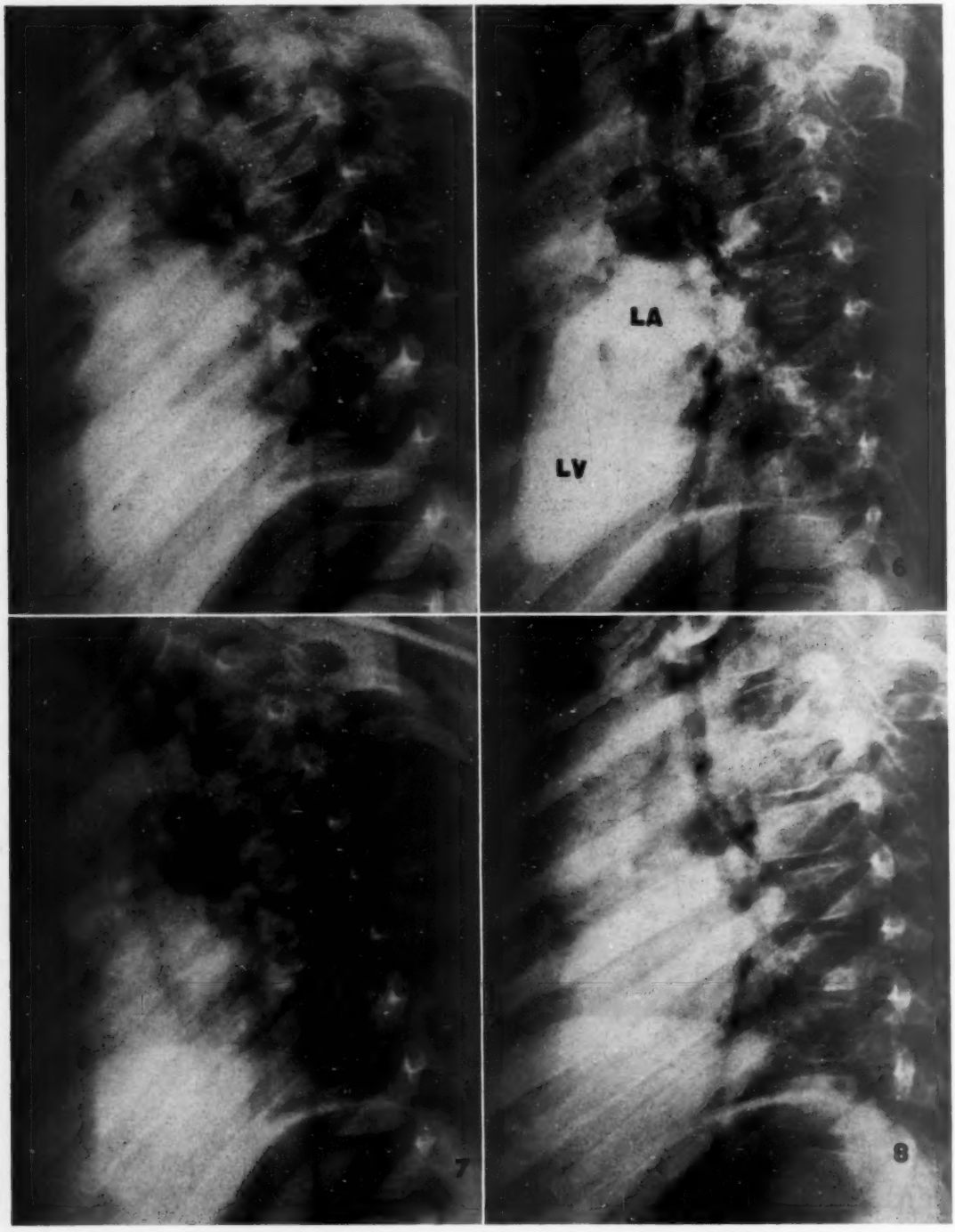

Fig. 9. Film 5. The opaque material has been injected into the aorta and is filling the major branches ascending in the neck.

Film 6. The left auricle and left ventricle and the aorta with its major branches are visualized at maximum density. Note the bulbus arteriosus and the coronary arteries below the letters *BA*.

Film 7. The density of the opaque material is beginning to fade out as it becomes diluted by the blood.

Film 8. Practically all of the diodrast has left the heart while a little remains in the aorta.

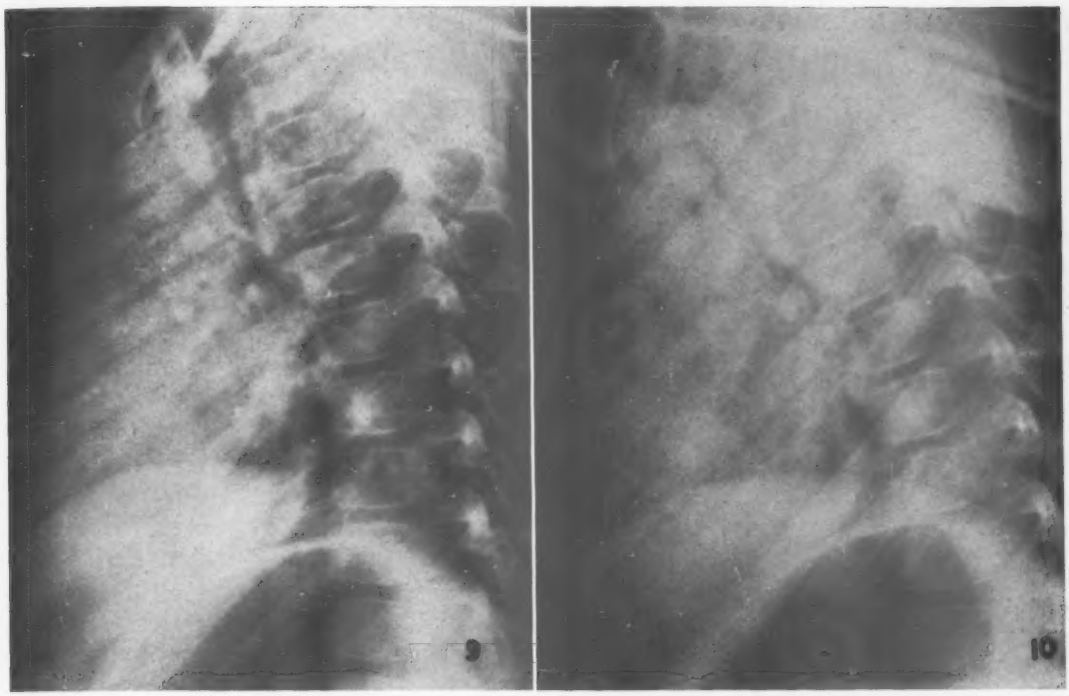

Fig. 10. Film 9. All the diodrast has passed into the systemic circulation and none remains in the cardiopulmonary circuit.

Film 10. A small amount of the opaque medium is returning from the systemic circulation into the superior vena cava. It is not sufficiently dense to be of clinical significance.

tion and by giving the younger children and infants a little sugar water in a bottle for them to suckle on during the examination.

CONCLUSIONS

(1) The tautograph offers a new principle in rapid serial roentgenography by utilizing a chain conveyor system for standard cassettes, by the incorporation of a self-cocking Potter-Bucky grid, and by the complete automatization of the entire equipment.

(2) Rapid serialization of roentgen-ray exposures by the full automatization of the equipment makes angiocardiography a practical technique in the diagnosis of infants and children with congenital heart disease.

(3) The technique has also been used to advantage in cerebral angiography.

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PULSATING EXOPHTHALMOS DUE TO DEFECTIVE DEVELOPMENT OF THE SPHENOID BONE

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UNILATERAL pulsating exophthalmos due to defective development of the bony wall of the orbit is a rare condition. Martin and Mabon[5] do not mention this cause in a "review of all reported cases," 812 in all, of pulsating exophthalmos. The condition has characteristic features and its recognition obviates the possibility of misguided treatment. The words of LeWald[4] may appropriately be quoted: "The recognition . . . is of extreme importance in roentgen diagnosis.

"In the first case which I observed in 1926 a previous erroneous diagnosis of sarcoma of the orbit with destruction of the orbital bony structures had been made, and the advisability of enucleation of the eyeball had been discussed. In a somewhat similar case reported by Rockliffe and Parsons actual removal of the orbital contents preceded by ligature of the carotid artery was performed and was followed by the death of the patient."

The condition was first described clearly by Dandy.[3] His patient was an intelligent girl of sixteen, who had had pulsating exophthalmos from birth. Roentgenograms showed that the posterior half of the roof of the orbit was missing. Dr. Arnold Knapp of New York, after a study of the roentgenogram, had suggested that this was a congenital defect. During a reparative operation, Dandy found the outer surface of the brain to be covered by large pools of fluid in the subarachnoid space; the leptomeninges were opaque and greatly thickened. The posterior two-thirds of the orbital roof was absent. There was no anterior clinoid process. The bony defect was covered by dura, which was firmly attached to the eyeball. The tip of the temporal lobe was covered by tremendously dilated subarachnoid spaces, continuous through the sylvian fossa with similar spaces over the frontal and parietal lobes. A large anomalous vein lay medial to the dura on the inner side of the middle cranial fossa. It represented the extradural cavernous sinus and was formed by the confluence of two large veins, the superior and inferior orbital veins, which pierced the dura covering the orbital contents. The carotid artery was not seen. Dandy inserted a piece of the outer table of the skull of proper shape, curvature and size into a tunnel between the dura and the orbital contents, so that it rested on bone on each side and in front. The patient benefited from the operation. Slight downward displacement of the eyeball remained, but there was no exophthalmos or pulsation of the eyeball. There was no diplopia five months after operation, although it is to be remembered that the vision of the left eye was poor (6/60) before operation and, after operation, the eye was reported to deviate downwards and inwards.

LeWald[4] described 4 cases (including that described by Dandy) with pulsating exophthalmos, in which roentgenography of the skull disclosed absence of the roof and posterior walls of the orbit. The anterior clinoid process was absent on the affected side. The orbit was enlarged, and its inferior wall depressed, so reducing the size of the maxillary antrum. The vision was variably affected. In 2 of the 4 cases there was evidence of diffuse neurofibromatosis, and 1 had other developmental defects, namely abnormal cervical vertebrae and an arteriovenous fistula of the leg. LeWald reviewed the literature and found several cases, described under varying diagnoses, which he considered to be examples of this condition. Of much interest were 4 cases reported by R. F. Moore,[7] in which pulsating exophthalmos was associated with diffuse neurofibromatosis. This association

prevented a clear statement of what was undoubtedly in the author's mind—that the bony defect was, in reality, a developmental one. Referring to a pathological report of a case by Rockliffe and Parsons, Moore stated that he suspected that the bone had never developed. In describing the pathological examination of this case, which was associated with a large plexiform neuroma of the orbit, Rockliffe and Parsons[9] reported that "the crista galli and central dividing crest of the parietal bone were pushed nearly one inch to the left of the median line. The whole of the orbital plate of the frontal, the lesser and greater wings of the sphenoid, and the lacrymal plate of the ethmoid were missing, and the inner margin of the supra-orbital ridge was serrated. The roof of the orbit was represented only by thickened dura mater. . . . " It is also interesting to note that, in a discussion of this case when it was originally shown by Rockliffe, Clarke[2] stated that he had demonstrated a patient in 1894, whose history left little doubt that the lesion was a developmental defect.

Anderson,[1] in a monograph entitled "Hydrophthalmia or Congenital Glaucoma," devoted a section to the association of congenital glaucoma with other anomalies, including von Recklinghausen's disease. He mentioned a number of authors who had described cases in which pulsation of the eyeball due to a defect in the orbital wall was also present, sometimes together with "elephantiasis of the lids." In some of these cases there was prominence of the temporal region and enlargement of the sella turcica. Apparently in most cases the condition was thought to be due to erosion of bone by a neurofibromatous mass, but some authors appear to have suspected that the coexistence of the two conditions was rather due to the tendency of development defects to occur together. More recently, A. E. Moore[6] described 2 cases in which neurofibromatosis was associated with proptosis and a defect of the orbital wall. Wheeler,[11] in describing the cases which he had seen in conjunction with LeWald, states: "Pulsation of the eyeball may depend upon communication between the cranial and orbital cavities, but pulsation probably does not occur unless a large part of the roof of the orbit is absent. Such a large defect may be (a) a congenital anomaly, an arrest of development, (b) an accompaniment of plexiform neurofibromatosis of the orbit (von Recklinghausen's disease) and in this condition the bony defect may be congenital, (c) a result of surgical removal of the roof of the orbit. Pulsation may be lost in a quantity of neurofibrous tissue between the orbitocranial communication and the dislocated eyeball."

CASE REPORT

A Warrant Officer, aged thirty-seven, stated that since he was fourteen years of age he had noticed "slight enlargement and slight movement" of his left eye. He suffered no inconvenience until three years before when working in a hot dusty atmosphere he developed conjunctivitis, which persisted for two years in spite of treatment. Some months after the onset of the conjunctivitis he began to suffer from intermittent pains which seemed to start in the center of his eyes, and extend through to the back of the head. The pain was worse on the left side than on the right. It became more frequent and severe during a period of hard work some two and a half years after its onset. The pain was of gnawing quality, and was not influenced by posture. Although the pain did not throb, he was conscious of pulsation, which was especially rapid when he was excited.

The left eyeball was prominent and depressed. There was deliberate backward and forward movement of the left eyeball, synchronous with the pulse. The amplitude of this movement with each pulse beat was slightly less than 1 mm. There was also a slower and less obvious variation, synchronous with respiratory movement and having an amplitude of about 0.75 mm. Compression of the jugular veins caused further protrusion of the eye. Compression of the left carotid artery lessened the amplitude of the pulsation, while bilateral compression abolished it. The eyeball was readily displaced backwards by pressure on the closed eyelids. The eye sank deeply into the orbit, beyond a normal position, until eventually con-

siderable discomfort resulted. No bruit was audible. The fundi were normal. The retinal veins on the left side were of the same diameter as those on the right. The visual acuity of each eye was good and equal and the visual fields were normal. The skull was of brachycephalic type, the left temporal region being more prominent than the right.

Roentgenological examination of the skull showed slight enlargement of the left orbit with depression of the inferior wall, the maxillary antrum being smaller on this side (Fig. 1). The left orbit had an appearance of uniform translucency in axial films, and, in contrast to the normal side, no shadows were seen corresponding to the orbital plate of the frontal bone, the lesser wing of sphenoid, or the orbital plate of the greater wing. The zygomatic process of the frontal bone and the frontal process of the zygomatic bone were wider on the left side. There was a large frontal extension of the posterior ethmoidal cells, and the ethmoidal cells medial to the left orbit were narrow. The left lateral wall of the sphenoidal sinus was closer to the median line than the right. An area of thinning was seen in the frontal bone above the lateral part of the zygomatic process. Lateral views (Fig. 1*B* and 5*B*) showed a large pituitary fossa, with a straight anterior wall. The orbital plate of the frontal bone terminated 7 mm. behind the ethmoidofrontal recess. A normal optic foramen with a large pneumatized anterior clinoid process was seen on the right side, while on the left side the corresponding structures were absent (Fig. 2). Roentgenograms of the base of the skull showed normal foramina ovale and spinosum on the right side. On the left side there was a single large foramen which opened widely into the foramen lacerum.

The initial pressure of the cerebrospinal fluid was 170 mm. Bilateral jugular compression caused a rapid rise to 550 mm., with a rapid fall on release of pressure. Pressure on the right jugular vein alone caused a rise to 480 mm., while pressure on the left caused a rise of only 50 mm. Pressure on the left eye caused a rise of 25 mm. within two seconds, followed by a slower rise to 380 mm. in ten seconds. Further pressure at this stage produced considerable discomfort which, on each occasion, necessitated release of pressure. The pressure of the fluid fell rapidly to the original level when the pressure on the globe was released. The rise of pressure was undoubtedly due to an increase in the bulk of the intracranial contents. The cellular and chemical constituents of the fluid were normal (the protein content amounting to 20 mg. per 100 cc.).

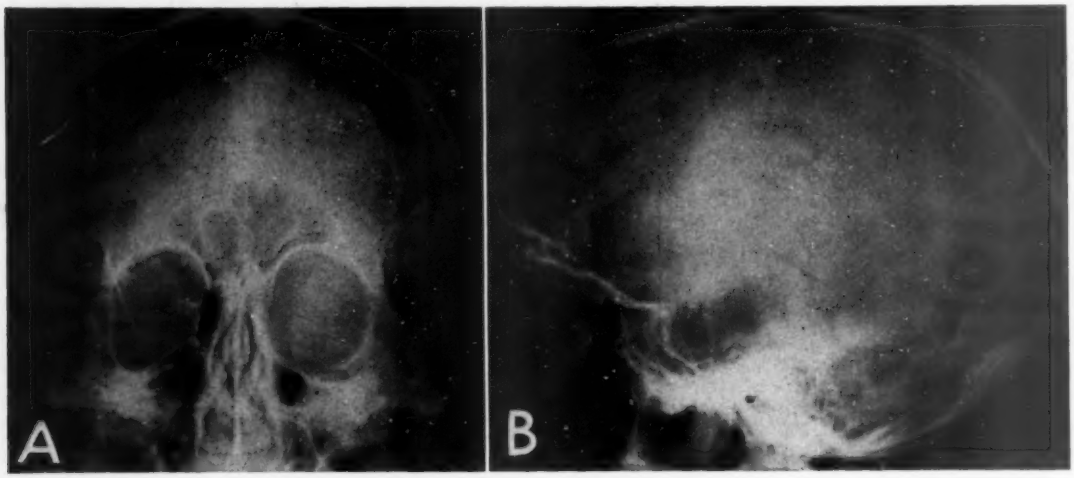

FIG. 1. Posteroanterior (*A*) and lateral (*B*) roentgenograms showing the absence of the roof of the orbit and the posterior orbital wall on the left side.

Encephalography was performed in an endeavor to obtain further information. The lateral ventricles were slightly enlarged. The right anterior horn was larger than the left, and the tip of the left temporal horn was considerably larger than the right. The septum pellucidum was situated to the left side of the midline. The cerebral sulci were wider than normal. A collection of gas was seen in the left orbit. In the anteroposterior view (Fig. 3*A*) it was seen below

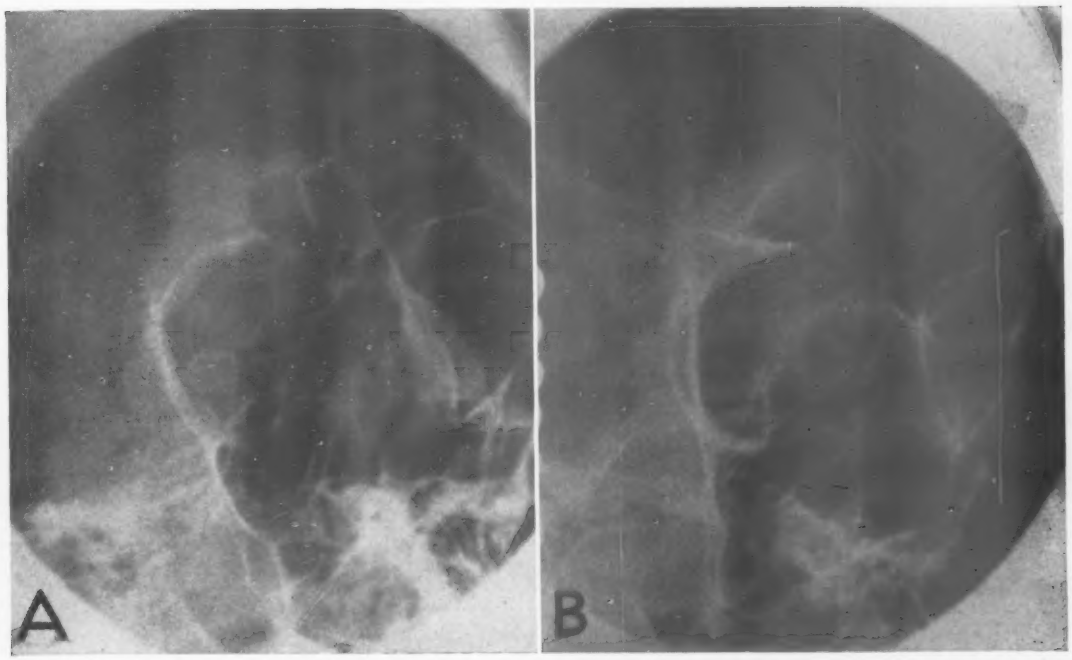

Fig. 2. Orbital films to show the optic foramen on the right side (*A*) and its absence on the left (*B*).

the level of the superior orbital margin, extending across the width of the orbit. The upper part of the shadow was indented, apparently by the convolutions of the inferior surface of the frontal lobe. A lateral film (Fig. 3*B*) showed the gas to extend forward to the posterior surface of the eyeball. These findings suggested the presence of an orbital encephalocele, due to defective development of the orbital wall. Subsequent inquiry from the patient's mother elicited the fact that the deformity had been noticed soon after birth. On the day after en-

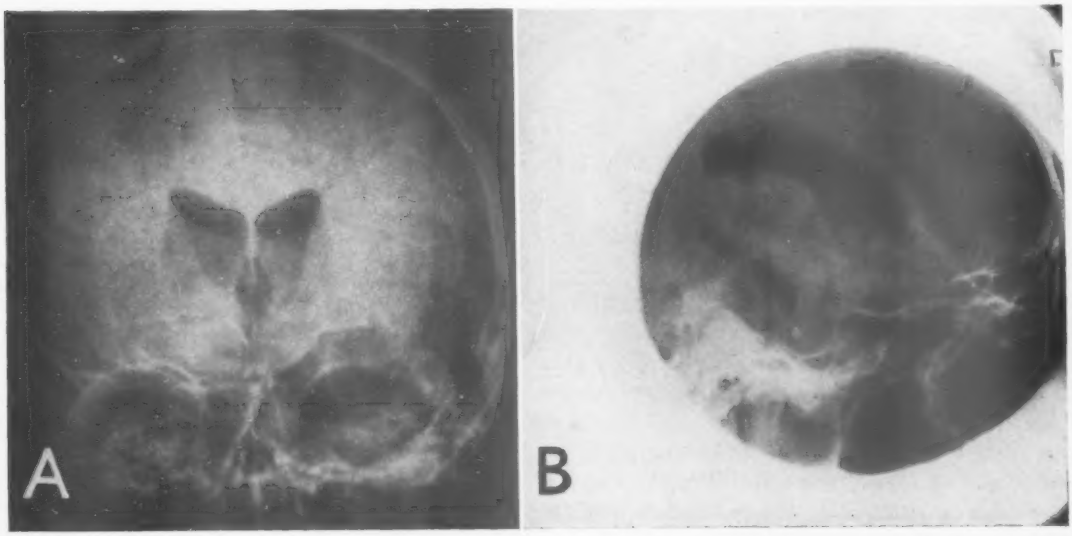

Fig. 3. Anteroposterior (*A*) and lateral (*B*) films taken with the brow up after a limited replacement of cerebrospinal fluid with oxygen. The subarachnoid space on the left side is seen to extend into what is normally the orbit. The large pituitary fossa and the defect in the roof of the orbit is seen in (*B*).

cephalography the left eye was less prominent than the right, although it still pulsated, and there was troublesome diplopia. Jugular compression temporarily restored the exophthalmos. The eye gradually became more prominent, although at the end of ten days the proptosis remained less pronounced than before encephalography.

Laminagrams showed that the floor of the middle cranial fossa was lower on the left side than on the right, especially anteriorly (Fig. 4).

FIG. 4. Laminagram in the coronal plane of the right clinoid process. The left clinoid process is absent. The asymmetrical body of the sphenoid, the depression of the floor of the middle fossa and the bulging of the temporal fossa are seen. The pterygoid processes are wider apart on the left side and the medial process more slender.

On the left side this part of the floor (formed by the greater wing of the sphenoid) turned upwards into the temporal surface without the broadening caused by the confluence of the orbital portion, which could be seen on the right side. Asymmetry of the sphenoidal sinus was evident, as was the absence of the anterior clinoid process on the left side. Sphenoidal cells extended into the base of the lesser wing on the right side and into the right anterior clinoid process. The pterygoid processes appeared to be more delicate on the left side than on the right. More posteriorly, on the left side, the foramen ovale could not be separated from the large foramen lacerum. In more anterior sections, the roof of the orbit was seen to be absent on the left side. The lesser wing of the sphenoid on the right side was unusually delicate. The floor of the ethmofrontal recess and of the left frontal sinus was thin. Laminagrams taken parallel to the sagittal plane showed the deformity in contrast to the conformity of the normal side (Fig. 5). It will be seen that the posterior part of the orbital plate of the frontal bone, the lesser wing of the sphenoid and the vertically directed orbital part of the greater wing were absent.

Finally, it may be stated that the patient's headache disappeared during his stay in the hospital and has not returned since. He states that his eye is "smaller and more balanced than before." In the absence of discomfort it seemed unnecessary to suggest operative treatment. A further deterrent was the possibility of inducing diplopia, which might persist if the proptosis were abolished.

DISCUSSION

The accepted descriptions of ossification of the sphenoid bone afford no clue to the pattern, and the constancy of pattern, of the defect. The greater wing, including the lateral pterygoid lamina, is said to ossify as a whole. The lesser wing and the medial pterygoid processes have separate centers, and all unite with the body which develops from multiple centers. It is possible that, in reality, the orbital plate has a separate center of ossification.

The pituitary fossa is abnormal in size and shape in all the recorded cases; sometimes it is described as being deep, sometimes wide and shallow. It seems likely that this is due to maldevelopment of the fossa, rather than to an enlargement of the pituitary gland. No gas passed into the fossa during encephalography, but this does not exclude the presence of a large diverticulum of the subarachnoid space. The defective orbital plate of the frontal bone may be of primary significance, indicating that the defective development is due to some regional cause affecting all the bones in this region. On the other hand, it is more probable that the orbital plate fails to develop because of the absence of the lesser wing of the sphenoid, with which it articulates. The bulging temporal fossa, which is a constant finding, may be due to the absence of the retaining tie provided by the lesser wing of the sphenoid and the orbital plate of the frontal bone.

The dural membrane rests in equilibrium

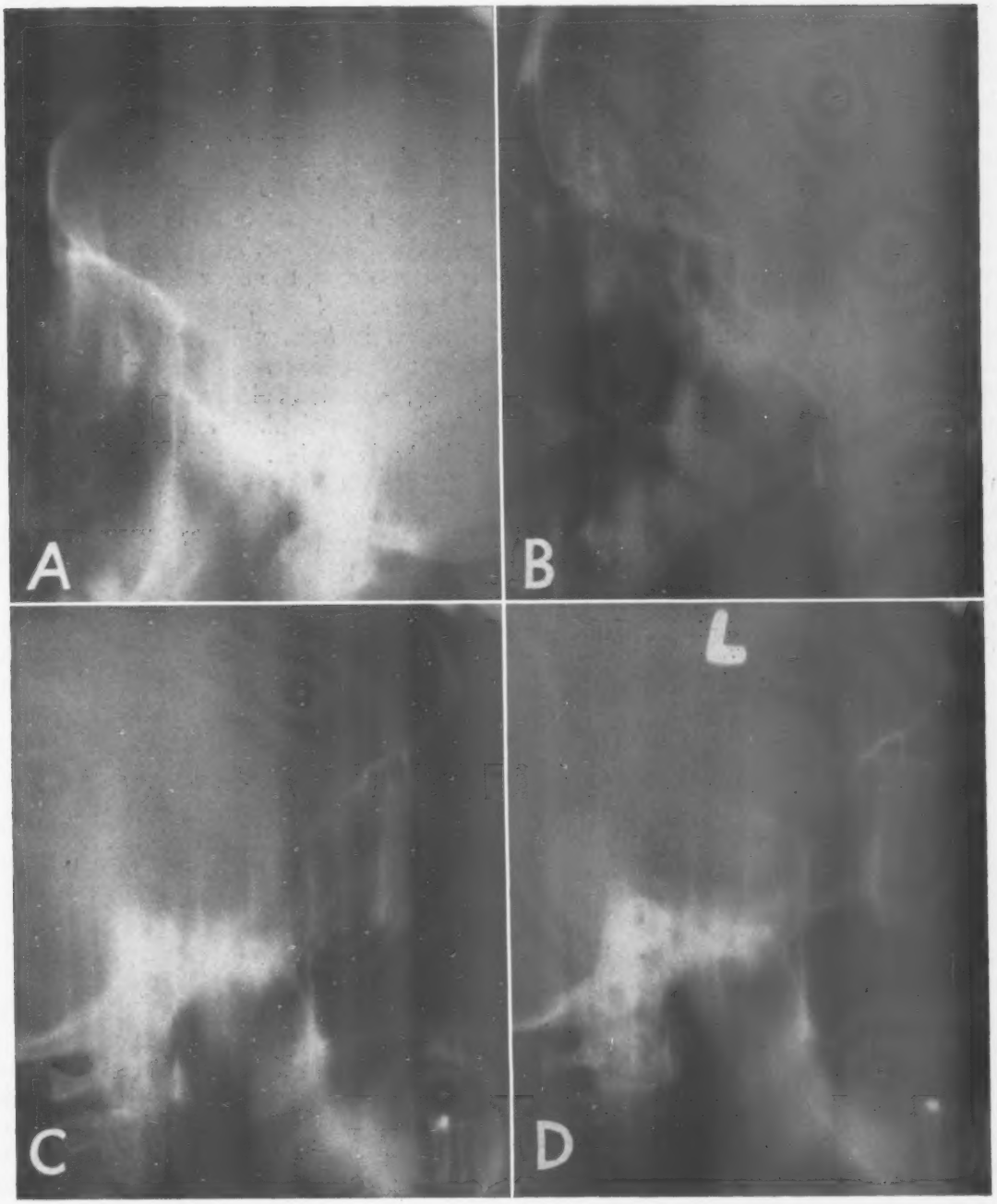

Fig. 5, *A*, *B*, *C*, *D*. Laminagrams in the sagittal plane. The contrast between the right (*A*) and left orbits (*C* and *D*) is well seen. A midline film (*B*), showing the deep sella turcica, is included for contrast.

when the pressure inside is equal to that outside. The pulsation is a visible expression of the fluctuation of the intracranial pressure. O'Connell[8] has calculated that the fluctuations due to cardiac and respiratory activity probably each lie between 20 and 60 mm. of fluid. Pulsation would be reduced were the dura mater tightly stretched over the aperture, particularly if the latter were small, or if an inelastic block of tissue existed between the dura and the eye. The first factor is probably sufficient to

explain the slight or absent pulsation after operative removal of the roof of the orbit, and the last, when neoplastic tissue intervenes. Pulsation has been observed in the dura after enucleation of the eye following an extensive comminuted fracture of the roof and posterior orbital wall. If the roof and posterior wall of the orbit are absent, the dura is able to prolapse into the orbit, for it no longer extends over two sides of a triangle, with a right angle bend at the lesser wing of the sphenoid (Fig. 5). The laxity of the dura under these conditions permits the transmission of the intracranial pulsation to the orbit.

Under the title "Pathogenesis of Intermittent Exophthalmos," Walsh and Dandy[10] describe a condition characterized by pronounced and rapid protrusion of one eye when venous stasis is induced by obstruction of the jugular veins. The eyeball may pulsate, and progressive failure of vision, pain and diplopia occur. Although they found descriptions of 111 cases, the cause had previously remained undiscovered. Exploration disclosed a mass of coiled vessels in the region of the sphenoidal fissure and the anterior part of the middle cranial fossa. They suggested that enlarged veins drained the aneurysm through the orbit, which itself was free of aneurysm. The size of these vessels would vary with the freedom of flow of blood through the jugular veins. Obstruction would cause distention of the large veins within the orbit, which would immediately cause proptosis. They considered that this was the only condition which could produce intermittent exophthalmos. It is interesting to note that enophthalmos, giving place to proptosis on jugular compression, existed in the patient described after encephalography. However, this was a transient state of affairs, occurring only when the intracranial pressure was artificially lowered. The presence of enophthalmos for some days after encephalography suggested that the fall of pressure was due to escape of cerebrospinal fluid through the puncture in the spinal membranes. It is interesting to note that the patient suffered no headache at this time (in contrast to the severe headache experienced when a fall of pressure follows lumbar puncture), probably because of the compensation effected by retraction of the orbital contents.

The condition is so characteristic that a mistake in diagnosis is unlikely, once the condition is known. The absence of distressing symptoms, the slow deliberate type of pulsation and the absence of bruit differentiates it from unilateral exophthalmos due to arteriovenous aneurysm. In the latter, pulsation is less marked and there is usually marked dilatation of the retinal and neighboring veins.

The result of the developmental defect may be properly termed orbital meningocele and encephalocele, but it differs from the better known condition to which these terms usually are applied. In the latter, the roentgenological picture is entirely different, for the sac usually escapes from the cranium through a gap between the cribriform plate of the ethmoid and the orbital plate of the frontal bone, and protrudes into the orbit between the latter and the lamina papyracea of the ethmoid. As the defect is usually situated anteriorly in the orbit, the eye is displaced laterally and downwards but is not unduly prominent.

The writer desires to express his gratitude to Major-General S. Roy Burston, Director-general of Medical Services, Australian Military Forces, for permission to publish this paper. Colonel D. J. Thomas, Commanding Officer, 115 (Heidelberg) Military Hospital, personally provided the facilities for taking the laminagrams.

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SOLITARY TUMORS OF THE CHEST*

THE DIFFERENTIAL DIAGNOSIS IN FIFTY PROVED CASES

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THE number of tumors of the chest coming to the attention of roentgenologists has increased since the beginning of mass surveys. These cases have been selected only to the extent that the roentgenologist reporting the first film study was unable to differentiate between two or more of the following tumors: *teratomas*, *aneurysms*, *intrathoracic thyroid*, *carcinoma* (primary or metastatic), *one of the lympho blastomas*, and so forth. Although the tumor was discovered in 9 patients of this group during a survey, no attempt was made to arrive at an exact diagnosis until a complete roentgenographic study had been carried out.

The purpose of this study was to determine which of the diagnostic studies offer the most help in arriving at an exact diagnosis in patients who present a solitary tumor of the chest.

STUDIES COMMONLY MADE

A. Roentgenoscopy of the Chest. This important diagnostic study should be employed routinely. The vocal cords should be studied both while at rest and during phonation. The detection of a paralyzed left vocal cord in a patient who has a mass along the left anterior mediastinum is important. Aneurysms of the arch of the aorta frequently cause a paralysis of the left recurrent laryngeal nerve. If the mass is above the aorta and is associated with a paralysis of a vocal cord, there is great likelihood that we are dealing with an invasive neoplasm. A tumor associated with an elevated leaf of the diaphragm, with paradoxical motion, is most likely involving the phrenic nerve.

An attempt should be made to separate the mass from the heart and great vessels. Rotation of the patient, while roentgenoscopic observations are made, will frequently make this differentiation possible. In some instances it will not be possible to confirm or rule out the possibility of a mass having originated in the heart or aorta.

The presence or absence of pulsation of the mass should be observed. The significance of these pulsations is questionable. Pulsations have frequently been accepted as presumptive evidence of the mass being an aneurysmal sac. This has resulted in numerous errors. I believe pulsation of the aneurysmal sac to be the exception rather than the rule. The most vigorous pulsations are noted when a solid tumor rests against a normal aorta. A diseased dilated aorta containing a thrombus is rarely capable of exhibiting vigorous pulsations, due to the inelasticity of its walls. Palpation of an accessible aneurysm frequently fails to reveal pulsations. In both of the innominate artery aneurysms in this series, careful repeated examinations by several examiners failed to detect the presence of pulsations. In some patients it is difficult to determine whether the mass is within the lung or in the pleural space. Close observation of the filling and emptying of the adjacent lobe will usually make this distinction possible. A mass which appears to be against the posterior chest wall might be displaced forward by air-containing lung behind it, if it is within the lung. The presence or absence of obstructive emphysema might also aid in this differentiation.

Studies of the barium-filled esophagus frequently give information concerning the exact location of the mass.

B. Roentgenographic Study. In addition to the conventional ventral and lateral projection films, a Potter-Bucky film is

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usually made. This assures a more adequate penetration of the mass, and affords us more accurate information concerning the presence or absence of calcium, uniform density, erosion of the adjacent bones, and so forth. If the mass is along the course of the esophagus, we have found films with the esophagus outlined by barium to be of considerable help.

All of the roentgenograms have been reviewed with particular reference to the following points:

(a) *Bone erosion.* None of the aneurysms showed evidence of bone erosion. In instances of erosion of the spine or of the sternum by a contiguous mass suspected of being an aneurysm, the diagnosis is relatively simple. Two of the nerve tissue tumors were of the "collar button" variety with one mass situated in the thorax and the other within the neural canal. They were joined by a narrow neck through an intervertebral foramen. In neither instance was there evidence of widening of the foramen or erosion of the adjacent bony structures, as observed by the roentgenograms and at exploration. Some of the large nerve tissue tumors rested against the posterior rib cage and dorsal vertebral bodies but there was no evidence of erosion observed at the time of operation or on the films. One of the tumors of nerve tissue origin determined to have undergone sarcomatous degeneration was later discovered to have metastasized to a vertebral body.

(b) *Presence of calcium.* The presence of calcium when in the form of bones or teeth establishes the tumor as belonging to the teratoma group. Relatively few teratomas show a calcium deposit. In the present group of cases calcium was found in 4 instances (carcinoma of the thyroid, wall of a bronchiogenic cyst, a thymoma and a neurofibroma with sarcomatous degeneration), and in none of the cases was it of help in establishing the diagnosis. When calcium is deposited within the walls of an aneurysmal sac it helps in establishing this diagnosis but in most aneurysms calcium cannot be detected.

(c) *Location of the mass.* It is commonly said that tumors of nerve tissue origin are found in the posterior part of the thorax but not anteriorly. We have found the neurofibromas in both the anterior and posterior parts of the thorax. Both of the ganglioneuromas were in the posterior gutter against the spine. Aneurysms are usually located anteriorly on the right and posteriorly on the left. In the present group of cases we have tabulated both exceptions. We find the serologic and neurologic findings of greater help in confirming or ruling out the diagnosis of an aneurysm than the location of the mass. One of the thymomas was so far from the normal thymus gland site that the diagnosis seemed unlikely but sections made from all parts of the tumor substantiated the diagnosis.

(d) *Effect on the adjacent structures.* The lack of bone erosion was mentioned above. Tracheal compression has been found due to carcinomas and aneurysms. Apparently this is due to the normal fixation of the aortic arch and innominate artery so that compression rather than displacement of the trachea occurs. The carcinomas which invaded the mediastinum also fixed and compressed the trachea. In several instances fixation of the trachea was detected by the bronchoscopist before there was roentgen evidence of compression. The teratomas, cysts, Hodgkin's disease, lymphosarcoma, tumors of nerve tissue origin and thymomas did not produce tracheal or vascular compression even when situated adjacent to the trachea and great vessels.

(e) *Pleural effusion.* Free fluid in the pleural space was found in 2 patients. In 1 patient 2,000 cc. of grossly bloody fluid was removed. At a subsequent exploration a spherical tumor (benign neurofibroma) was readily removed. The second patient whose roentgenograms appeared identical showed clear fluid with an abundance of tumor cells. Subsequently pleural metastases were detected.

C. Bronchoscopy. This diagnostic measure which contributes so much in most pulmonary diagnostic problems affords

little help in tumors of this nature. The detection of a paralyzed vocal cord, fixation of the trachea, compression of a bronchus, and so forth, have value in determining the operability of the tumor but afford little positive diagnostic information. In only 2 cases of this group was the bronchoscopist able to secure a satisfactory specimen for histologic study.

D. Sputum Study. The detection of hair or sebaceous material is of great value in establishing the diagnosis of a dermoid. The presence of neoplastic cells is of value in establishing the diagnosis of a malignant tumor. We have used this study in only a few cases of this series but there is evidence to suggest that a more routine search for the presence of tissue cells is warranted.

E. Planigraphy, thoracoscopy and lipiodol bronchography have not afforded us help in the differential diagnosis of these tumors.

F. Histopathologic Study of Aspirated Pleural Fluid Cells. Pleural fluid was present in 2 of these cases. In 1 patient who had clear fluid a definite diagnosis of adenocarcinoma was made. The other patient had 2,000 cc. of *bloody fluid* removed and this revealed no abnormal cells. At a subsequent exploration an 8 centimeter spherical tumor was removed from the pleural space and was found to be a neurofibroma. There was no evidence of neoplastic changes and the tumor was not adherent. The blood vessels over the capsule of this tumor were profuse and the area showed changes indicating the site of the bleeding. This patient had a history of intermittent attacks of pleurisy for seven years. Bloody pleural fluid is not always indicative of a malignant tumor of the pleura and should not contraindicate exploration.

G. Response to Irradiation. A test of response to the effects of irradiation is of value in some of the patients in whom a diagnosis cannot be made by aspiration biopsy, or in whom a tumor of the lymphoblastoma series cannot be excluded. The disadvantages are: (1) the length of time that must elapse before conclusions can be reached (two to three weeks), (2) malignant tumors of a resistant type cannot be excluded by this method. Benign tumors are not influenced by the irradiation. We have given a course of irradiation to masses, ultimately determined to be aneurysms, without any harmful results to the patient. This test is of practical use in some of those masses which can be definitely localized to the mediastinum.

H. Hormone Assay. The Aschheim-Zondek or similar tests have not been employed. It was thought that in those cases where the differential diagnosis included a teratoma, a negative hormone report would not be conclusive and a positive hormone report would be dependent upon the complete exclusion of any other source of the hormone.

I. Artificial Pneumothorax. Advances in chest surgery have made this procedure unnecessary as a preliminary phase of preparation for surgical exploration of the thorax. It does not appear to have value except in those few cases where it is not possible to determine whether a mass is within the lung or adjacent to it in the pleural space or mediastinum.

J. Aspiration Biopsy. The value of this method of procuring material for histopathologic study is no longer questionable. The technique employed is essentially that outlined by Martin and Ellis[1] in 1934. In any group employing this method, the success depends upon the degree of cooperation of the surgeon, the radiologist and the pathologist, rather than upon differences in technique. In our group the aspiration is carried out under biplane roentgenoscopic guidance in order to be certain of the exact site of the needle during the aspiration maneuver.

In solid tumors of the types reported here, a negative report has little significance unless repeated on three or four occasions, and then due consideration must be given to the possibility that we are dealing with a non-neoplastic process. It is our opinion that teratomas, cysts, carcinomas, sarcomas, aneurysms, and the lym-

[1] Martin, H. E., and Ellis, E. B. Aspiration biopsy. *Surg., Gynec. & Obst.*, 1934, *59*, 578–589.

phoblastomas can be confirmed or ruled out with a high degree of accuracy by needle aspiration biopsies.

We have no hesitancy in inserting a needle of small caliber into a mass so situated that an aneurysm must be considered a diagnostic possibility. In these cases a very thin needle is used. There have been no complications and no diagnostic errors as determined by subsequent exploration or autopsy.

Teratomas have been aspirated without infecting the pleural space or the development of any other complication.

On a number of occasions we have attempted to pass a needle into small, spherical masses of the type commonly referred to as tuberculomas. In these instances we have been unsuccessful. Several times the mass was contacted but the needle would not penetrate it.

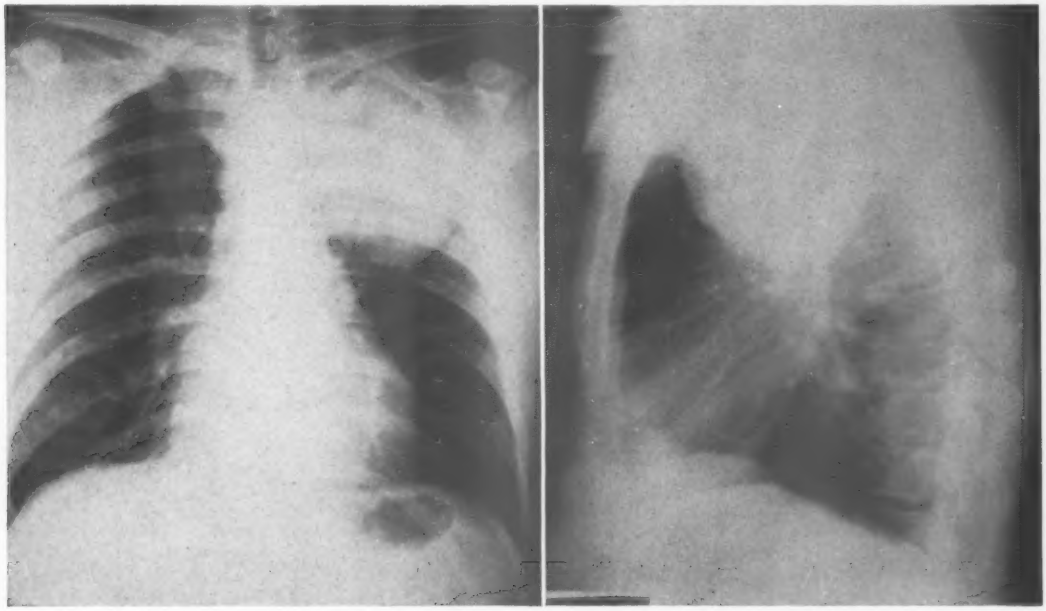

Fig. 1. Bronchiogenic carcinoma. This paramediastinal mass resembled a cyst. It was within the lung and did not involve the parietal pleura. Aerated lung surrounds the mass on its ventral, lateral and dorsal borders (Table 1, Case 1).

DISCUSSION OF CASES

A. *Carcinoma of the Lung.* These tumors were of two types: (1) Bronchial carcinomas originating adjacent to the mediastinum and which were oval or spherical in outline (Fig. 1). (2) Primary spherical parenchymal tumors. In most instances it was not possible to determine roentgenographically whether the mass originated in the mediastinum or adjacent to it. The primary spherical parenchymal tumors were identical with blood borne metastatic lesions. The fact that only one tumor was present and that it was relatively large suggested that we might be dealing with a primary neoplasm.

B. *Tumors of Nerve Tissue Origin.* These tumors were of two types: (1) Neurofibroma or neurinoma which develop from the nerve sheaths and occur anywhere along the course of a nerve. They are usually benign but, at times, they undergo transformation and become invasive tumors such as sarcomas. They are usually oval in contour with the long axis along the longest diameter of the body. They may occur in any part of the chest where nerve tissue is found but most of them occur posteriorly along the dorsal spine. (2) Ganglioneuromas which develop in the

TABLE I

TABULATION OF CASES—CARCINOMA OF LUNG

Age and Sex	Clinical Features	Location of Mass	Size in cm.	Needle Biopsy	Treatment and Subsequent Course	Diagnosis and Remarks
Case 1 M—58 W.M.	Cough for 4 weeks	Left paramediastinum lung root to apex	10×12×14	Positive	Pneumonectomy. Uneventful	Bronchiogenic carcinoma
Case 2 M—44 M.T.	Pain left upper chest, 4 months	Left paramediastinum lung root to apex	11	Positive	Exploratory. Inoperable. Retrogressive	Carcinoma. Slight response to complete course of deep roentgen therapy
Case 3 M—52 E.R.	Pain in right chest and arm. Weight loss, 7 weeks	Right paramediastinum lung root to apex	9	Positive	Inoperable. Horner's syndrome. Pain in arm. Unimproved	Undifferentiated carcinoma
Case 4 M—61 W.B.	Cough with hemoptysis, 3 weeks	Lung root to apex, right paramediastinum	9	Not done; tissue by bronchoscopy	Exploratory. Inoperable. Unimproved	Squamous cell carcinoma bronchus Grade 2
Case 5 M—56 C.F.	Cough, hoarseness, pain in left chest for 9 months	Left paramediastinum lung root to apex	11	Positive	Inoperable due to left recurrent laryngeal paralysis	Squamous cell carcinoma bronchus
Case 6 M—50 H.S.J.	Chest pain, cough with hemoptysis. Weight loss, 4 months	Right paramediastinum anteriorly lung root	8×6×6	Positive	Exploration. Inoperable due to mediastinum involvement. Progressively downhill. Death	Undifferentiated cell carcinoma grade 4, with invasion of mediastinum
Case 7 F—57 M.B.	Cough with hemoptysis, 2 months	Left paramediastinum lung root to apex	7	Positive	Pneumonectomy. Uneventful	Squamous cell carcinoma, bronchiogenic
Case 8 M—52 V.A.	Hoarseness, headache and backache, 4 months	Right paramediastinum lung root to apex	10×9×7	Positive	Inoperable, left recurrent laryngeal palsy. Roentgen therapy. Unimproved under roentgen therapy	Carcinoma squamous cell type. Death in few months
Case 9 M—54 R.P.	No complaints in chest. Found in survey	Right paramediastinum posterior lung root to apex	7×6×5	Positive	Pneumonectomy. Uneventful	Grade 4 undifferentiated cell carcinoma
Case 10 M—64 H.L.	Cough with hemoptysis. Hoarseness. Weight loss, 5 months	Right paramediastinum lung root to apex	8×8×6	Positive	Inoperable. Laryngeal nerve paralysis and fixed trachea	Grade 4 undifferentiated cell carcinoma
Case 11 M—48 E.L.	Cough with hemoptysis. Hoarseness and weight loss, 2 months	Left paramediastinum lung root to apex	12	Positive	Inoperable. Laryngeal nerve and phrenic paralysis. Fixed trachea	Carcinoma
Case 12 M—33 E.D.	Cough. Dyspnea on exertion. Hoarseness, 4 months	Left paramediastinum lung root to apex	6	Positive	Inoperable. Roentgen therapy. Left vocal cord paralysis. Death in 6 months	Carcinoma
Case 13 F—35 H.W.	Cough with hemoptysis, 3 months	Left upper lobe parenchyma	3×5×6	Not done; tissue by bronchoscopy	Bronchoscopic removal (partial). Seen recently. Treatment not yet completed	Adenoma bronchus
Case 14 F—35 M.McL	Survey film. No complaints	Right upper lobe parenchyma	4	Positive	Pneumonectomy. Uneventful	Adenoma bronchus, low grade malignancy. The removed specimen and the material obtained on aspiration were identical
Case 15 M—45 J.G.	Cough, fatigue, fever, 5 months	Left upper lobe. Parenchymal lingula	6	Negative. Surgical biopsy	Pneumonectomy. Unimproved	Bronchiogenic carcinoma, squamous cell Grade 3. Died 1 year later cerebral metastases
Case 16 F—63 A.Y.	Survey film. No complaints	Left upper lobe, parenchyma	5	Negative. Surgical biopsy	Pneumonectomy. Uneventful	Mucoid carcinoma. Needle biopsy repeated 3 times but insufficient material for diagnosis
Case 17 M—63 J.D.	Pain in chest. Dyspnea on exertion. Weight loss	Left base to anterior mediastinum	10×10×12	Positive	Had effusion partly covering mass. Had pleural involvement. Death in few months	Adenocarcinoma

cells of the peripheral nervous system, commonly in the sympathetic ganglia, may occur in any part of the thorax.

The symptoms of these tumors are chiefly due to local compression. Many are discovered accidentally (Fig. 2).

C. *Carcinoma of the Thyroid.* The carcinoma probably arose in aberrant thyroid tissue. There was no evidence that the mass developed in an intrathoracic exten-

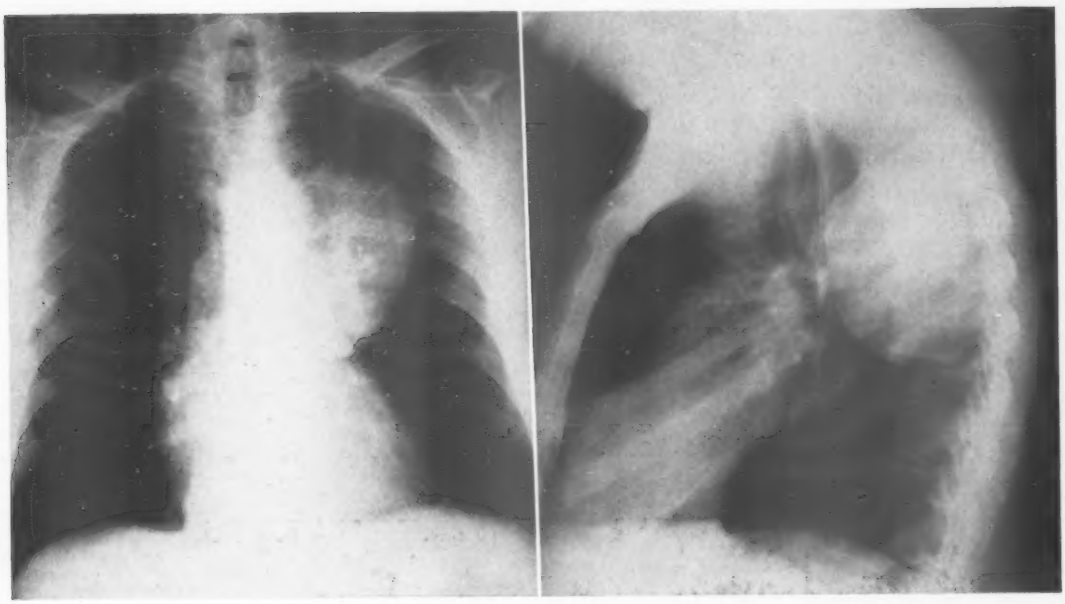

FIG. 2. Neurofibroma. This tumor is typical in appearance and location. The long axis of the tumor corresponds to the long axis of the body. It fills the gutter formed by the dorsal spine and the posterior rib cage (Table II, Case 1).

sion from the normal thyroid. The trachea had not been displaced in the usual manner of inthrathoracic or substernal goiters. The appearance and location of the mass suggested a tumor of nerve tissue origin (Fig. 3).

D. Bronchiogenic Cysts. These appear as solid tumors but are filled with fluid. They

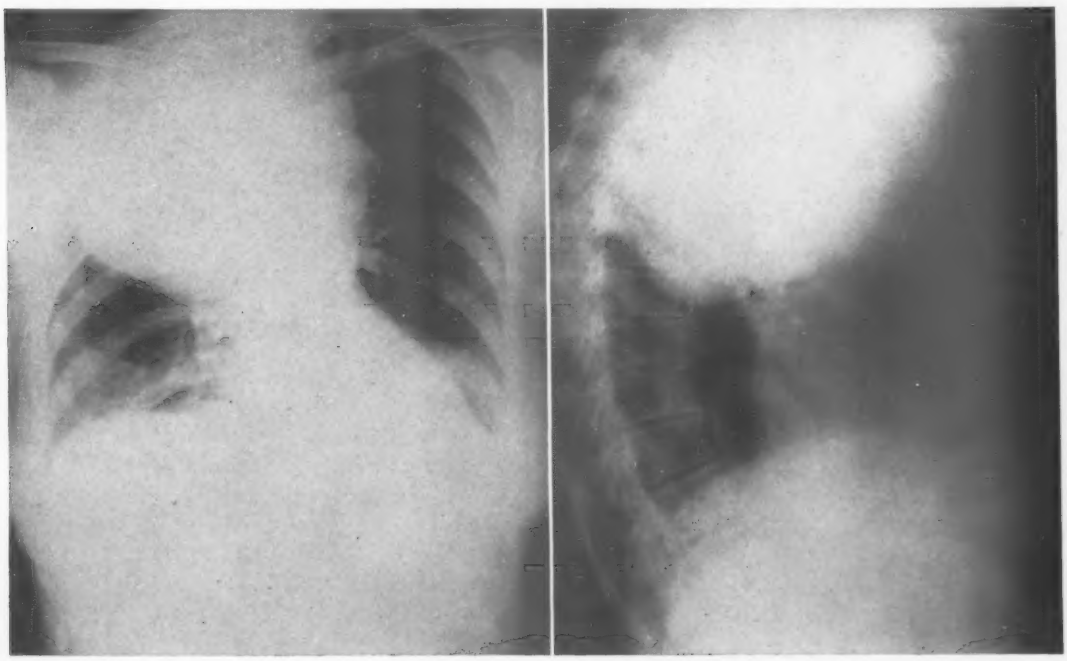

FIG. 3. Carcinoma of thyroid. The contour and location of the mass resembles tumors of nerve tissue origin (Table IV, Case 1).

are of embryonic origin but do not have the characteristics of teratomas. The cyst wall has the histology of a bronchus. It consists of fibrous tissue which contains lymphoid follicles and mucous glands. The lining consists of a thin layer of pseudostratified cuboidal and cylindrical cells. There is no histopathologic evidence of any inflammatory response. The contents are clear fluid mixed with mucus. There are no contents similar to that found in teratomas. There were two cysts of this type—one adjacent to the anterior mediastinum and the other adjacent to the posterior mediastinum simulating an aneurysm of the descending aorta (Fig. 4).

E. Aneurysms of the Innominate Artery. In both patients the aneurysm appeared separated from the aorta, which appeared normal. The trachea had been displaced to the left and it was compressed. The lateral view showed the oval outline of the mass extending anteriorly and posteriorly from the line of the trachea. A neoplasm was suspected in both instances as being more likely than an aneurysm. Aspiration of the mass by a small gauge needle established the diagnosis (Fig. 5). The second patient had a carcinoma of the stomach and the mass in his mediastinum was thought to be a metastatic process.

F. Aneurysms of the Aortic Arch. In most instances it is possible to differentiate between mediastinal tumors and aneurysms. The location of the mass in relation to the normal aorta is helpful but there are often exceptions. In 3 instances the diagnosis was

TABLE II

TABULATION OF CASES—TUMORS OF NERVE TISSUE ORIGIN

Age and Sex	Clinical Features	Location of Mass	Size in cm.	Needle Biopsy	Treatment and Subsequent Course	Diagnosis and Remark
Case 1 M—72 S.G.	Cough, dyspnea, weight loss, 6 months	Left posterior gutter lung root level	13×10×10	Positive	Unimproved	Neurofibroma. General condition of patient contraindicated surgery
Case 2 F—22 E.F.	Pain left chest, 20 months	Left lung root area	10×12×12	Not done. Surgical biopsy	Excision. Uneventful	Benign neurofibroma
Case 3 M—58 W.C.	Cough and pain in chest, 2 months	Left posterior mediastinum gutter lung root level	16	Positive	Roentgen therapy. Unimproved	Neurofibroma. Inoperable due to spine involvement. Sarcomatous degeneration. Death in few months
Case 4 M—44 R.C.	Unable to walk. Loss of bowel and bladder control, 30 years	Left posterior mediastinum gutter, lung root to apex	7.5×6×6	Not done. Surgical biopsy	Excision. No improvement	Neurofibroma. No improvement during 6 months follow up. Cord compressed by mass—"collar button" type
Case 5 M—45 W.B.	Pain and numbness in right shoulder. Difficulty in walking, 11 weeks	Right D-7, D-4 gutter	20×8×7	Not done. Surgical biopsy	Laminectomy. Excision. Death later	Neurofibroma. Collar button" type tumor. Confirmed at autopsy
Case 6 F—10 G.V.	Cough and fatigue, 4 years	Right posterior apex gutter	10×11×13	Not done. Surgical biopsy	Excision. Death	Ganglioneuroma
Case 7 F—28 M.K.	Pleurisy, intermittent attacks for 7 years	Right base, posterior mediastinum to gutter	8	Not done. Surgical biopsy	Excision. Uneventful	Neurofibroma. Had bloody effusion. Blood came from vessel in capsule as determined from removed specimen
Case 8 F—51 E.M.	Cough, dyspnea, pain in chest for 18 months	Right base, front to back	18	Negative	Excision. Uneventful	Neurofibroma
Case 9 M—41 C.M.	Pain in chest, 5 years	Left lung root anterior mediastinum	12×12×13	Not done. Surgical biopsy	Exploration. Retrogressive	Neurofibroma. Invaded mediastinum and lung. Specimen removed, histopathologically benign. Death in 3 years
Case 10 M—22 A.W.	No complaints. Detected in survey film	Left posterior mediastinum to gutter, lung root to apex	10×12×16	Negative	Excision. Uneventful	Ganglioneuroma
Case 11 M—60 C.H.	Cough, hemoptysis, dyspnea, pain and hoarseness for 2½ years	Right anterior mediastinum lung root to apex	15×18×20	Negative. Surgical biopsy	Exploratory. Unimproved. Roentgen therapy	Neurofibroma with sarcomatous degeneration Inoperable due to invasion

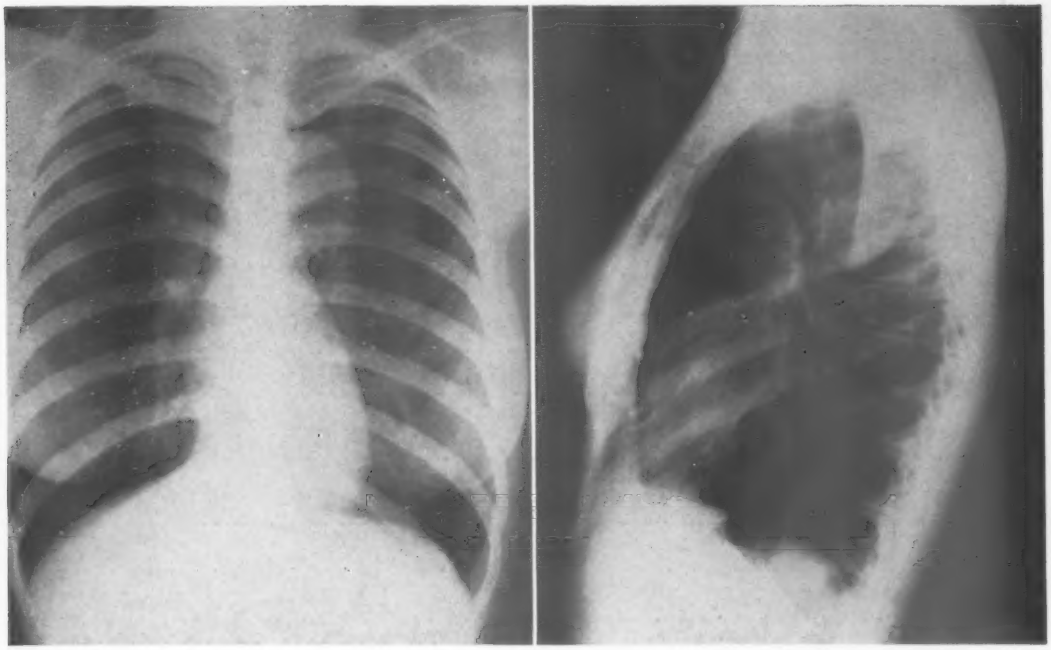

Fig. 4. Bronchiogenic cyst. This mass was superimposed on the aorta and could not be distinguished from it (Table IV, Case 7).

in doubt due to the unusual position of the mass particularly in its relation to the trachea and esophagus (Fig. 6).

G. *Teratomas.* There is nothing characteristic about these tumors. A few dermoids have teeth or osseous structures pres-

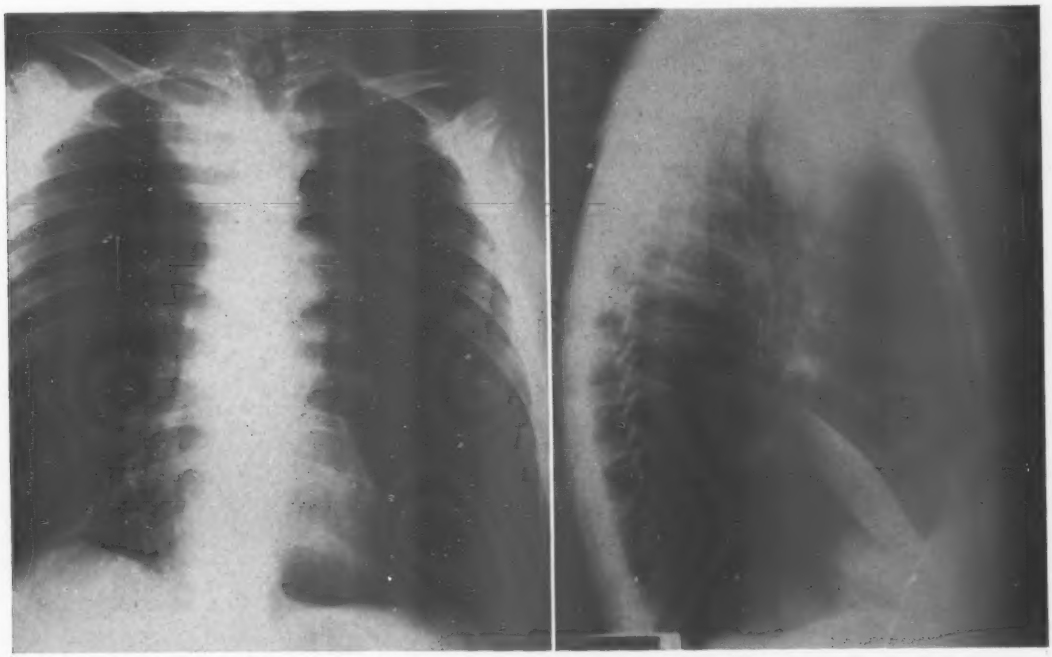

Fig. 5. Aneurysm of innominate artery. It was not possible to differentiate between an intrathoracic goiter, a paramediastinal bronchial carcinoma and an aneurysm. The marked compression of the trachea can be seen. Atelectasis of the anterior basal segment of the right lower lobe is also shown (Table III, Case 4).

TABLE III

TABULATION OF CASES—TUMORS WITHIN MEDIASTINUM

Age and Sex	Clinical Features	Location of of Mass	Size in cm.	Needle Biopsy	Tissue (How obtained)	Treatment and Subsequent Course	Diagnosis and Remarks
Case 1 M—11 T.C.	Pain in back for 3 years	Behind heart midline at base	11×7×5	Positive	Lymph node biopsy later	Roentgen therapy. Death 4 years after onset	Hodgkin's disease. Mass appeared to be a dermoid or neurofibroma
Case 2 M—16 N.S.	Cough and fever for 3 weeks	Right lung root	12×8×6	Not done	Lymph node biopsy later	Roentgen therapy. Death 1 year after onset	Hodgkin's disease. Mass appeared to be vascular or a teratoma. Trial roentgen treatment made differential diagnosis
Case 3 F—28 H.D.	Cough and fever for 6 months	Right anterior paramediastinum	8	Not done	Autopsy	Roentgen therapy. Death 4 years after onset	Hodgkin's disease. Mass appeared to be a paramediastinal carcinoma. Marked response to trial dose roentgen therapy
Case 4 M—36 E.S.	Substernal pain and dyspnea for 2 months	Right paramediastinum	8×8×10	Positive		Antiluetic. Slightly improved	Aneurysm innominate artery, lues. Positive serology
Case 5 M—50 M.G.J.	Abdominal pain due gastric cancer	Right paramediastinum	10×8×6	Not done	Autopsy	Antiluetic. Death gastric cancer	Aneurysm innominate artery, lues. Positive serology
Case 6 M—52 H.B.	Brassy cough, dyspnea and hoarseness for 8 years	Right mediastinum	11	Not done	Autopsy	Splitting of sternum. Death due to asphyxiation. No rupture	Aneurysm of aortic arch which had extended behind trachea and esophagus and displaced them forward. Sac began 3 cm. from aortic ring
Case 7 M—70 C.G.	Cough and dyspnea for 4 months	Left anterior mediastinum	12×8×10	Not done	Autopsy	Antiluetic. Death due to rupture	Aneurysm of transverse aortic arch. Appeared on film identical with paramediastinal tumors. Had positive serology
Case 8 M—42 M.W.	Cough, dyspnea, and pain in chest for 18 months	Right mediastinum between trachea and esophagus	10	Not done	Autopsy	Antiluetic. Death due to rupture	Aneurysm of transverse and descending portion extending across midline between trachea and esophagus. Thought to be a cyst. Serology positive
Case 9 M—38 F.H.	None in chest	Right anterior mediastinum lung root	5×5×4	Positive		Roentgen therapy, very sensitive. Improved for 2 year follow up	Lymphosarcoma. Mass identical in appearance and location with the teratomas
Case 10 F—11 K.S.	Cough and wheezing for 6 years	Left mediastinum extending to base	10×12×15	Not done	Surgery	Excision. Uneventful	Thymoma. Easily removed
Case 11 M—58 J.McC.	Dyspnea	Left anterior mediastinum, lung root to apex	13×12×12	Positive	Surgery	Unimproved	Thymoma (malignant). Inoperable due to invasion
Case 12 M—53 E.F.	Pain in chest and fatigue for 18 months	Right anterior mediastinum ascending aorta level	9×5×3	Not done	Surgery	Excision. Death	Teratoma. Resembled aneurysm. Death resulted from bilateral pneumothorax and complications
Case 13 M—20 S.S.	Heart conscious. Pain in left shoulder for 3 weeks	Left anterior mediastinum lung root	10×8×7	Positive	Surgery	Excision. Uneventful	Teratoma, needle biopsy specimen adequate and representative

ent to give a clue but the solid tumors about the mediastinum are roentgenographically indistinguishable from any of several tumors of the type reported here. If the tumor is chiefly of ectodermal origin and if it communicates with a bronchus, hair or sebaceous material may be expectorated (Fig. 7).

H. *Thymoma.* There is nothing characteristic about these tumors, either as to location, contour, or effect on the adjacent structures. One of the tumors grossly resembled a lipoma. This mass was easily removed and had only a few small blood vessels running into it.

I. *Mycelia of Fungus.* In this patient the mass resembled a parenchymal carcinoma. Repeated needle aspirations revealed a core of tissue made up of mycelia of the aspergillus. The possibility that this is a secondary invader rather than the primary etiologic factor is considered as being most likely. There were some changes in the pleura about the mass suggestive of an old tuberculous process but the tubercle bacillus could not be found. The patient has

TABLE IV

TABULATION OF CASES—MISCELLANEOUS

Age and Sex	Clinical Features	Location of Mass	Size in cm.	Needle Biopsy	Tissue (how obtained)	Treatment and Subsequent Course	Diagnosis and Remarks
Case 1 F—60 S.B.	Dyspnea, pain in shoulder	Right apex, posterior mediastinum	11×12×14	Positive		Tracheotomy. Death —48 hours	Carcinoma Grade 4, thyroid. Mass appeared to be a nerve sheath tumor or cyst
Case 2 F—11 J.M.	Cough for 8 years	Left lung root	10×8×5	Negative	Surgery	Pneumonectomy.	Fibroma. Mass had extended along lung root vessels. Tumor benign
Case 3 M—45 J.K.	Dyspnea, heart conscious and difficult swallowing	Left posterior gutter behind heart	15×15×10	Not done	Surgery	Partial excision. Improved	Fibrosarcoma. Invaded mediastinum and was inoperable. Much of mass was removed
Case 4 M—59 W.E.	Cough for 9 years	Right upper lobe in parenchyma	8	Positive	Bronchoscopy	Unimproved	Mycelia of fungus. Roentgen appearance was that of a neoplasm
Case 5 M—21 M.L.	None. Found on survey	Left anterior chest adjacent to ribs	11×9×5	Not done	Surgery	Excision. Uneventful	Rhabdomyosarcoma
Case 6 F—68 A.B.	Dyspnea, weakness, weight loss	Left thorax	25×23×20	Negative	Autopsy	None due to lack of time. Death within 48 hours	Rhabdomyosarcoma
Case 7 F—20 D.H.	Complaints none. Found on survey	Left posterior mediastinum level aortic arch	6×6×7	Not done	Surgery	Excision. Uneventful	Bronchiogenic cyst
Case 8 F—39 F.W.	Wheezing and dyspnea	Right anterior mediastinum lung root level	11×13×16	Positive		Aspiration. Relieved. Sac remained	Bronchiogenic cyst
Case 9 F—52 M.V.	None. Found on survey	Right base anteriorly	23×15×12	Negative	Surgery	Uneventful	Fibroma. Films made 9 years ago were reported negative but a review of them shows that a tumor 7×5×3 cm. was present in the right middle lobe region against the anterior chest wall

been followed several years and there has been no change, so there is little likelihood that we are dealing with an underlying neoplasm (Fig. 8).

J. *Adenoma of the Bronchus.* This interesting tumor is usually detected after it has produced partial occlusion of a bronchus by its endobronchial component. We have been aware, for several years, that the bulk of the tumor is outside the bronchus in many cases. While some of these tumors are low grade carcinomas, others appear benign when detected and remain so. Of the two tumors of this type, one was spherical and was thought most likely to be a metastatic tumor (Fig. 9). There was no endobronchial growth on sections of the removed lung. The other patient's tumor appeared spherical and there was a mass projecting into the bronchus but it had not yet produced atelectasis.

TABLE V

RESULTS OF ASPIRATION BIOPSY

Diagnosis	Positive	Negative	Not Done
Carcinoma of the lung (all types)	13	2	2
Nerve tissue tumors	2	3	6
Carcinoma of the thyroid	1	0	0
Lymphosarcoma	1	0	0
Fibroma and fibrosarcoma	0	2	1
Bronchiogenic cysts	1	0	1
Aneurysms (all types)	1	0	4
Teratomas	1	0	1
Thymoma	1	0	1
Rhabdomyosarcoma	0	1	1
Mycelia of fungus	1	0	0
Hodgkin's disease	1	0	2
	23	8	19

K. *Hodgkin's Disease.* There were 3 patients who had tumors of this kind in whom Hodgkin's disease was not suspected at first. In 1 patient the spherical mass was situated adjacent to the mediastinum but it appeared to be within the lung and so resembled a bronchial carcinoma (Fig. 10). A second patient showed the tumor along the

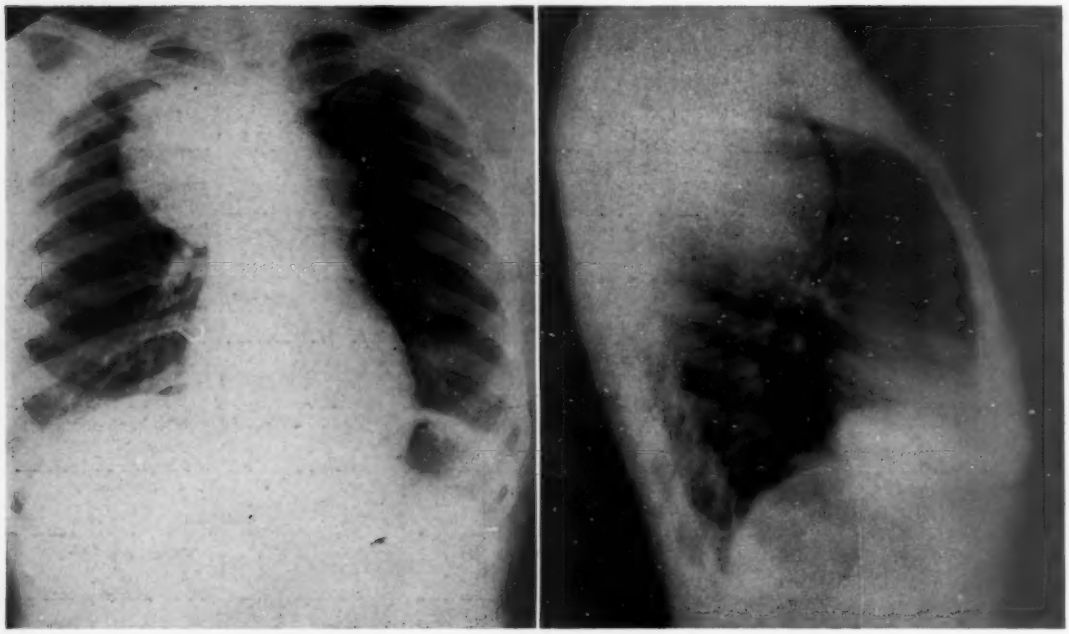

Fig. 6. Aneurysm of the aorta. The sac involves the transverse and first portion of the descending aortic arch. It extends to the right between the esophagus and trachea, pushing the trachea forward and compressing it (Table III, Case 8).

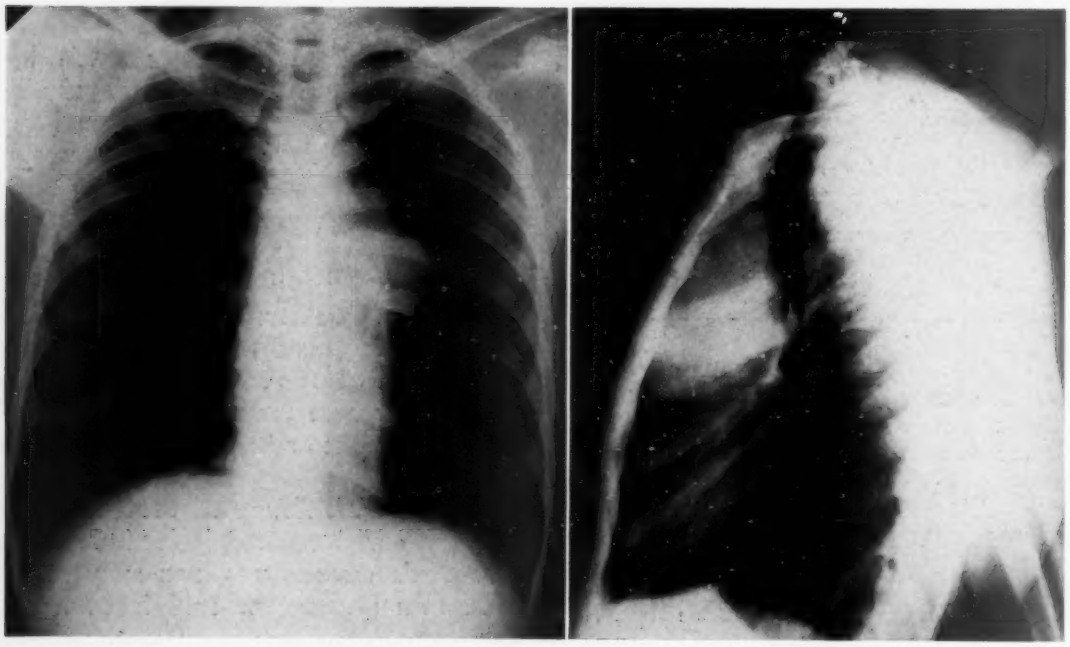

Fig. 7. Teratoma. This tumor was solid and made up of all three layers. There is no characteristic appearance of these tumors. Many errors are made if a presumptive diagnosis is arrived at by a study of the roentgenograms (Table III, Case 13).

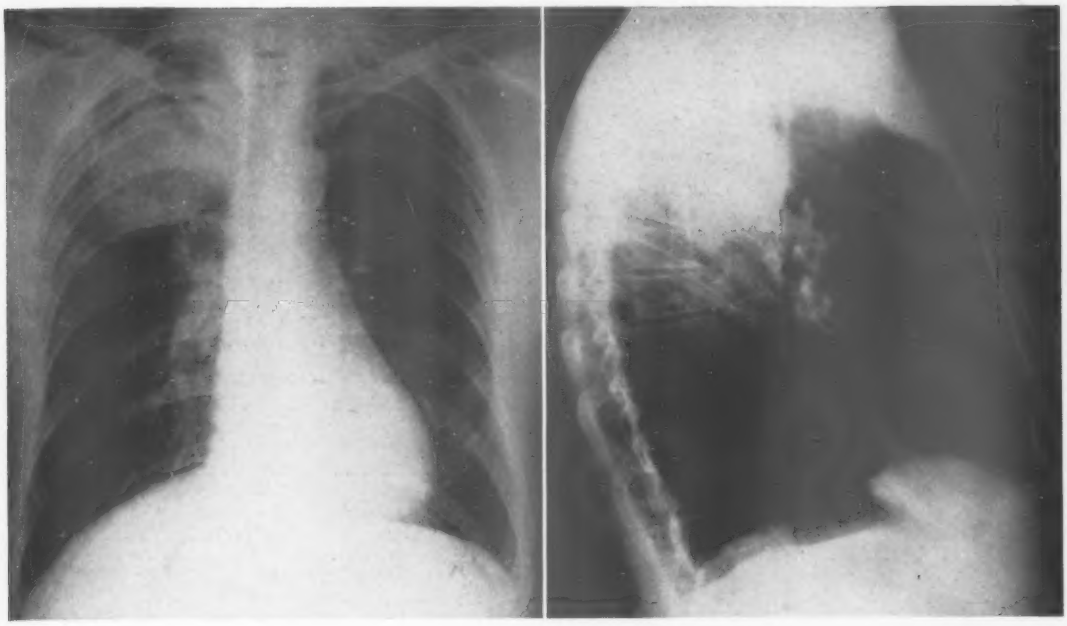

Fig. 8. Mycelia of fungus. This spherical mass was thought to be a bronchial carcinoma. The fungus was identified as aspergillus which was probably a secondary invader of an old tuberculous cavity (Table IV, Case 4).

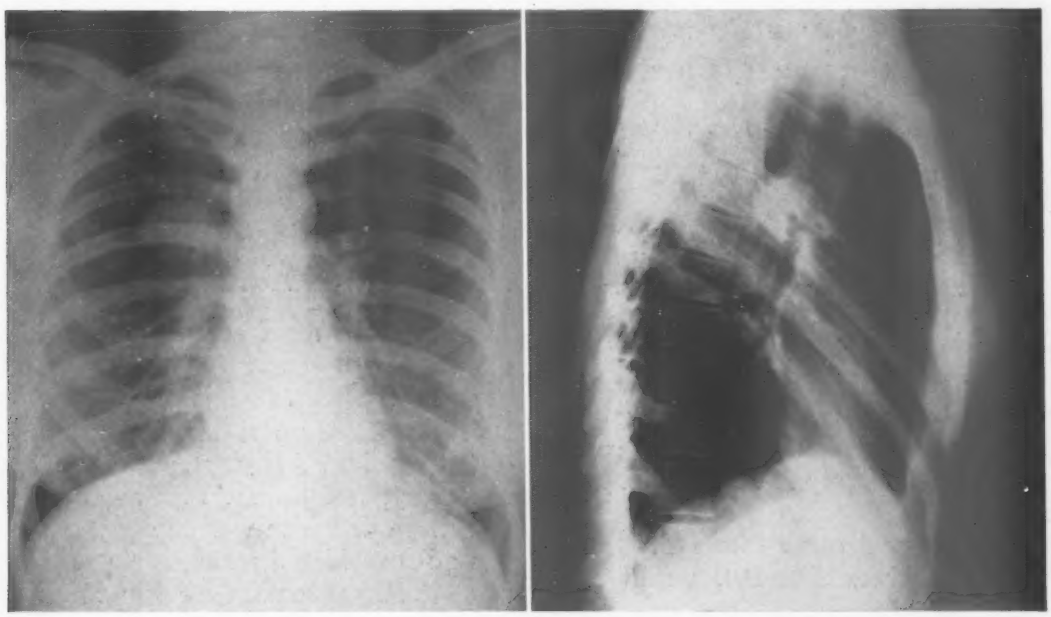

Fig. 9. Adenoma of the bronchus. This spherical tumor had not produced symptoms. There was no reaction in the adjacent lung. The tumor grew during a short period of observation. Histopathologically the tumor was a low grade carcinoma (Table 1, Case 14).

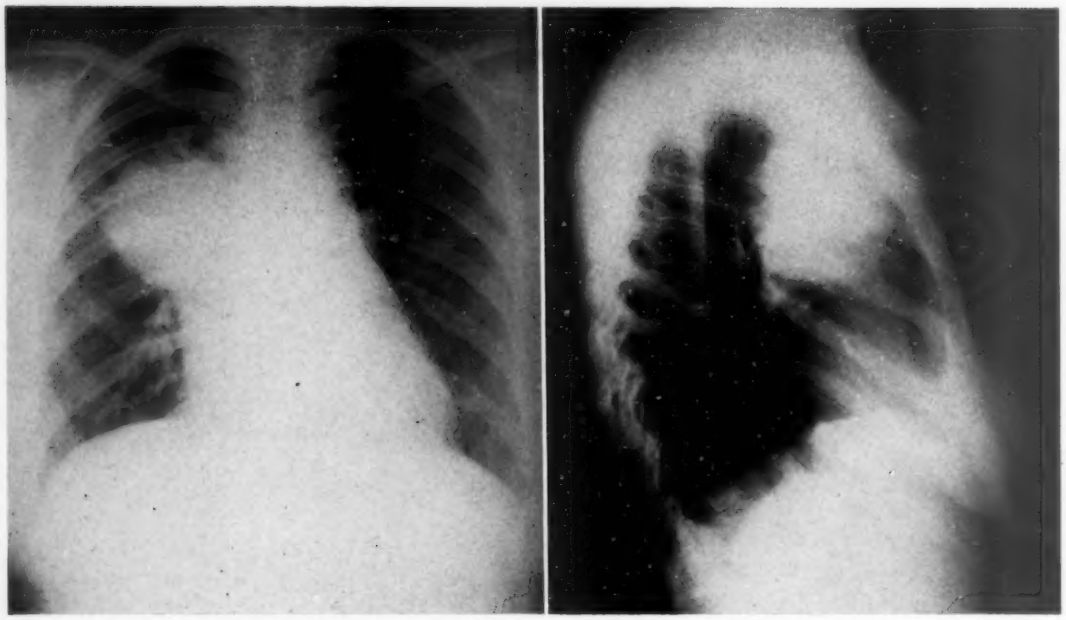

Fig. 10. Hodgkin's disease. This circumscribed paramediastinal mass presents a roentgen appearance nearly identical with a bronchial carcinoma and with an aneurysm which we have seen recently (Table III, Case 3).

ascending aorta and it appeared to be an aneurysm although a teratoma was also considered likely. In the third patient the tumor was behind the heart in the gutter against the dorsal spine and it appeared identical with the tumors of nerve tissue origin.

L. *The other tumors* of this group showed no gross characteristics that would in any way aid in their differentiation.

SUMMARY AND CONCLUSIONS

1. An increasing number of solitary tumors of the chest are coming to the attention of roentgenologists and thoracic surgeons as a result of mass surveys.

2. Competent roentgenologists are unable to make an accurate diagnosis by roentgenoscopy and a study of the films in many instances.

3. Fifty proved cases (those illustrated are representative) have been reviewed in order to determine the best means or arriving at an exact diagnosis. Most of these tumors are of three types: (a) bronchial carcinomas, (b) nerve tissue tumors, and (c) aneurysms.

4. Intelligent management of patients having solitary tumors of this kind (many of which were asymptomatic) depends upon the exact nature of the mass. It is a great advantage to be able to establish the diagnosis promptly so that therapeutic measures can be planned and carried out without delay.

5. Needle aspiration under roentgenoscopic guidance for the purpose of procuring material for histopathologic study afforded us the most prompt and accurate method of establishing the diagnosis (74 per cent of cases).

6. There are no roentgen findings that can be considered characteristic of any particular type of tumor. In many instances the diagnosis which was finally established was not considered among the possibilities when the first examination was made.

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BRONCHIAL PERISTALSIS*

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THE pulmonary parenchyma is in close relation to bronchi, vessels, septa, made up of connective tissue, and the pulmonary pleura. There is no portion of the pulmonary parenchyma of any considerable volume which does not contain bronchial branches. Thus any change in volume of any portion of parenchyma must cause simultaneous bending, twisting, lengthening or shortening of the contained bronchi. That these changes in the bronchi occur has been shown by means of bronchial casts made in expanded and collapsed lungs. The bronchoscopists were the first to see directly some of these changes in the living. Jackson reported that he was familiar with the rhythmical enlargement and diminution, elongation and shortening, twisting and bending of the normal bronchi observed through the bronchoscope. Due to the lesser respiratory changes in the central, hilar portion of the lung, and to the greater rigidity of the larger bronchi, these changes in length are minimal in the larger bronchial trunks during quiet respiration. The expansibility of the bronchi increases toward the periphery as the cartilaginous rings become thinner and are replaced by fewer and fewer cartilaginous plaques, while the muscular layers and elastic fibers grow thinner and scarcer. The elastic qualities of the respiratory bronchioles and alveolar ducts approach those of the lung parenchyma.

The classical anatomic description and the more recent studies by Baltisberger,[1] Macklin,[9,10] Miller,[12] and others, demonstrated the large amount of elastic and muscular tissue in the entire bronchial tree. These morphologic facts by themselves suggest functional plasticity of the bronchi. This was later proved in more detail by roentgenologic observations. Macklin (1925) analyzed the lengthening and shortening, the spreading and approximation to each other of the bronchial branches in relation to the eccentric anchoring of the lung at the hilum and the uneven expansion of the different parts of the thoracic cage.†

Heinbecker[4] (1927) observed the widening of the bronchi on inspiration, and their narrowing on expiration. This observation was confirmed by Hudson and Jarre[6] (1929) Huizinga[7] (1940), and others, and can be observed regularly during routine bronchography with roentgenograms taken in deep inspiration and expiration. These changes can be sufficiently explained by mechanical stress and strain exerted on the bronchial wall. There is little doubt that the lengthening of the bronchi is caused solely by the transmitted pull from the lung; the shortening is caused by the elastic recoil of the tissue, including the bronchial walls, since the bronchi do not carry any muscular fibers in longitudinal arrangement. The bronchial muscles are arranged in an approximately circular fashion, the "geodesic network" of Miller.[12] Therefore a synergic action of the bronchial muscles concomitant with mechanical widening and narrowing of the bronchi is a definite possibility.

Peristaltic movements of the bronchi were first described by Bullowa and Gottlieb[2,3] in 1919. In a brief note they described roentgenoscopic observations made on dogs, the bronchi of which were made visible by an oily barium suspension; they saw "a slow peristaltic wave of low amplitude which emptied the lungs without cough and without invoking ciliary movement."

† Macklin's careful analysis of the uneven expansion of different portions of the lung is correct though it was made originally on a mistaken premise. He took the normal hilar shadows and lung markings for the expression of the bronchial tree and compared plain roentgenograms taken in inspiration and expiration without injecting any opaque matter into the bronchi. As a matter of fact, there is very close parallelism in the arrangement of vessels and bronchi, except for the central hilar regions.

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Reinberg[13] (1925), first experimenting with a bismuth suspension in the trachea of the goose, saw wavy contractions and bending of the trachea, and stated "and this bow is slowly drawing upward." He continued, "The contrast substance, being lifted by peristaltic action upwards. . . ." He also observed "the characteristic picture of peristalsis in man. A bronchus of the third or fourth order very soon after the injection of bismuth, represented as the shadow of a cylinder or column, becomes elongated, twisting, protruding upwards, further redressing itself, even flexing downward. Now the regular-shaped band-like shadow narrows, and has a divided fragmental appearance, falls into a few columns or spindles; the isolated shadows walk upward, collect themselves in nodules, leaving a clear interspace, disappear, again appear —in short, the picture on the screen is animated, living. These movements proceed comparatively slowly, yet with such a speed that there is no reason to attribute them to the ciliary activity." Mayeda[11] interpreted his observations in a similar way.

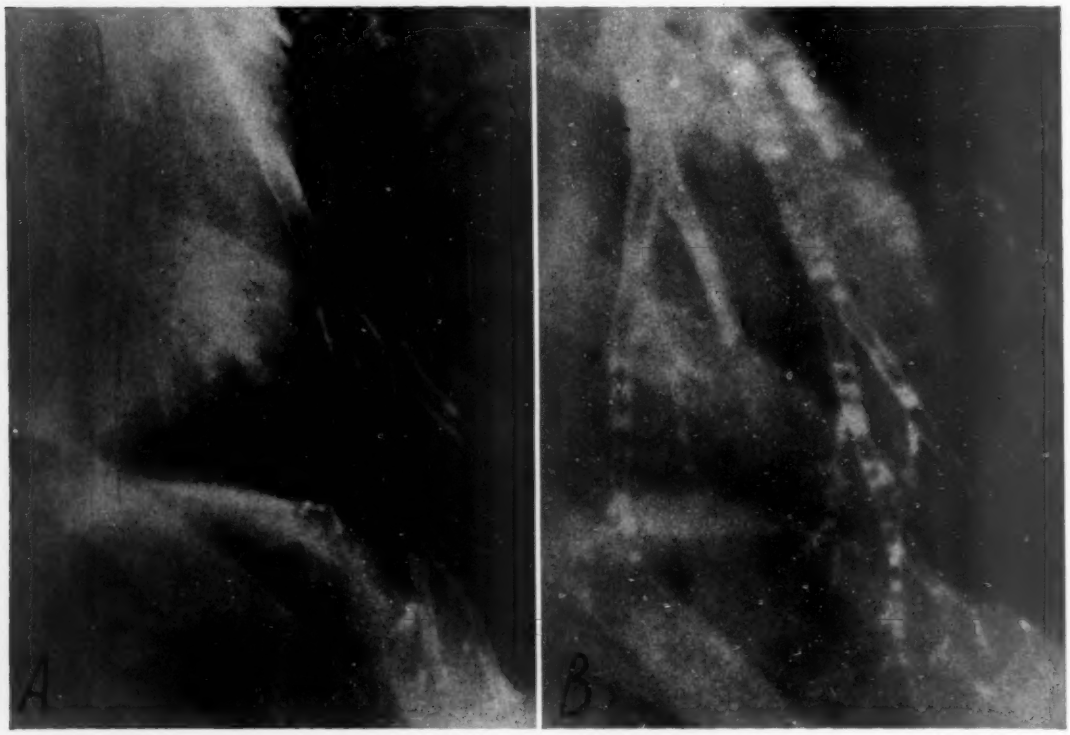

FIG. 1. Bronchograms. Reproductions in natural size showing a portion of the left lower lung field both in inspiration and expiration. *A*, inspiration; the bronchi are lined by a thin film of opaque oil (retouched). *B*, expiration; the same bronchi are readily identified. They are shorter and narrower. Plugs of oil form bridges from wall to wall. At their sites the lumen of the bronchi is narrower. The air-filled sections between oil plugs are patulous, but not contracted.

PRESENT OBSERVATIONS

In bronchographic roentgenograms, the bronchi of the fourth and fifth order of the lower lobes present themselves in three ways, depending upon the degree of filling and phase of respiration. (1) The bronchus is completely filled with opaque oil; it appears as a band with parallel smooth contours. I have never seen in healthy persons any narrowing suggesting a localized contraction or multiple evenly spaced narrowings suggesting a moving contracting wave. (In asthmatics cylindrical and more rarely annular narrowings are quite common.)

(2) There is just enough oil to form a thin film coating the bronchial wall while the lumen of the bronchus contains air. Here, also, the bronchial walls are perfectly straight and not deformed by any irregularity suggesting localized contractions. (Again with the exception of instances of asthma or bronchitis.) (3) Some bronchi contain air and oil irregularly alternating with each other in shorter or longer columns or droplets. This picture observed under the screen often shows a slow centripetal movement of the oil droplets. This phenomenon apparently seen by the early observers was interpreted as proof of a peristaltic action of the bronchi. We have found pairs of roentgenograms taken on the same person in inspiration and expiration, respectively, as most illuminating (Fig. 1, *A*, *B*). If the inspiration film shows merely lining of the bronchial wall, the expiration film shows the corresponding bronchi filled alternating with oil and air. In expiration the bronchus is shorter and narrower as can be measured or even seen with the naked eye. The amount of oil present in the bronchus inspiratory dilatation, sufficient to coat the wall, is crowded together on a smaller surface in the collapsed bronchus during expiration so that the layer becomes thicker and the oil forms bridges and plugs extending from wall to wall. At the site of many of those bridging plugs the bronchus appears to be narrower than in the adjacent air-filled sections. By looking at those roentgenograms, one gets the impression that surface tension is the controlling factor in this phenomenon. One should keep in mind the fact that the air and oil make visible the lumen and inner relief of the bronchus; they do not provide any further information about the bronchial wall as such. There is no necessity to assume that those ring-like narrowings represent a dent or bend throughout the entire thickness of the wall of the bronchus. The mucous membrane is loosely attached to and readily movable upon the deeper layers of the bronchial wall. In postmortem specimens of over-contracted lungs, one can often observe the mucous membrane of the bronchi arranged in transverse folds as a result of the heaping up effect which occurs when the bronchus shortens and narrows. Those roentgenograms suggest that the droplets of oil by means of their viscosity and surface tension elevate an abnormally large mucosal fold, thus making the bronchial lumen appear narrower. The slow centripetal motion of these droplets, as observed roentgenoscopically, should be interpreted as caused by the action of the ciliated epithelium.

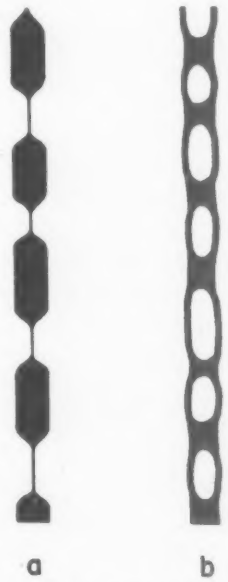

Fig. 2. (a) Diagram showing the manner in which the interrupted oil column was interpreted on the basis of roentgenoscopic observation solely. (b) The actual distribution of oil and behavior of the bronchus as revealed by roentgenograms.

The early observers, working with the poorer technical means at their disposal, apparently interpreted this phenomenon as illustrated by the diagram (Fig. 2*a*). The continuous oil column is broken up by segmental contractions separating individual oil droplets or spindles. The slow progressive motion of the oil droplets was thought to be due to the squeezing effect of those slowly advancing rings of contraction; i.e., a perfect picture of peristaltic waves.

With modern roentgenographic tech-

nique, we never see contraction between those oil droplets in healthy individuals. The bronchus, if patent, is lined with a film of oil and otherwise air-filled. The bronchial wall itself cannot be discerned on roentgenoscopy (the means of observation of earlier authors) (Fig. 2*b*).

We interpret the narrowing at the site of the oil plug as an effect of surface tension of the oil, causing the lifting of mucosal folds. There is no way of disproving that these narrowings are due to the localized annular contractions of peristaltic waves. If this interpretation was correct, these contractions might, by narrowing the bronchial lumen and approximating the walls, cause the oil film to flow together, thus forming plugs. These waves of contraction, while moving, would carry the oil droplets along by means of surface tension. There is, however, another fact that strongly militates against the conclusion that this phenomenon is due to peristaltic contractions: we never see constricting rings in bronchi completely lined with oil and otherwise air-filled. In this case one may object, there is no need for the expelling action of peristalsis and the body does not mobilize this defense mechanism. However, we never see these narrowings either, if the bronchus is completely filled with oil, and here an expulsive defense operation would be very much in need. The narrowing rings have been observed solely where air and oil mix in the bronchus, only in instances where the effects of surface tension between air and oil in addition to that between oil and tissue becomes operative. We therefore conclude that this phenomenon is not peristaltic in origin.

Though we generally think of peristalsis as progressive contractions of the fibers of a circular muscle layer, there is no doubt that the longitudinal musculature of tubular viscera has its part in the performance of peristalsis. There is lengthening and shortening of segments of the tubular viscus concomitant with the passage of the annular contraction. All other hollow viscera exhibiting peristaltic action have circular and longitudinal muscle systems. The smooth muscles of the bronchi are arranged solely in a somewhat modified circular fashion, the "geodesic system" of Miller. All students of the anatomy of the lung and bronchi agree in regard to the complete absence of longitudinal muscular systems.

This leads to the conclusion that the observed phenomena are probably due to surface tension and not to peristalsis of the bronchi. Since the theory of peristaltic activity of the bronchi was based solely on roentgenologic observations, according to our present knowledge, a further conclusion must be that nothing is known about bronchial peristalsis. The observations of earlier authors were correct; their interpretation was erroneous due to the incomplete facilities at their disposal twenty and more years ago.

SUMMARY

The assumption of peristaltic motility of the bronchi is based on the roentgenologic observation of the breaking up of opaque oil columns and slow centipetal motion of oil particles. These phenomena have been reviewed and interpreted as being due to surface tension and ciliary action. This observation does not permit any conclusion as to the occurrence of bronchial peristalsis.

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DOUBLE CONTRAST ROENTGENOGRAMS OF THE STOMACH

AN ADDITIONAL AID IN DIAGNOSIS

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IN an effort to improve our accuracy in the diagnosis of lesions of the stomach we have been using an air-filled balloon in the barium-containing stomach.

This is not an entirely new procedure but these exact means have apparently never before been described in roentgenological literature. The first mention of such a procedure is in 1915 by Rogers and Hardt.[1] They used a balloon coated with a mixture of bismuth subnitrate and vaseline and covered by a second balloon for observing the physiological movements of the stomach. They made no mention of diagnostic possibilities.

In 1941 D'Eloia[2] described, in a preliminary report, a Miller-Abbott tube with two balloons, one of which was inserted into the first part of the duodenum and filled with water as an anchor, then rugar was given. The two illustrations presented were excellent. This method required placing the end of the tube in the duodenum, which is often a problem, then filling one balloon with water and the other with air, all of which is quite time consuming.

Wasch and Epstein[3] in 1944 reported the inflation of the stomach with air through a stomach tube after a contrast meal but used no balloon. They visualized tumors of the fundus but had little control over the positioning of the air or its retention.

Thomas[4] in 1945 used air alone in the stomach to demonstrate large tumors and abscesses about the pancreas but he makes no reference to the diagnosis of intrinsic lesions of the stomach.

Poppel and Roach[5] in 1945 describe the use of effervescent powders with an opaque meal. Tumors of the fundus were demon-

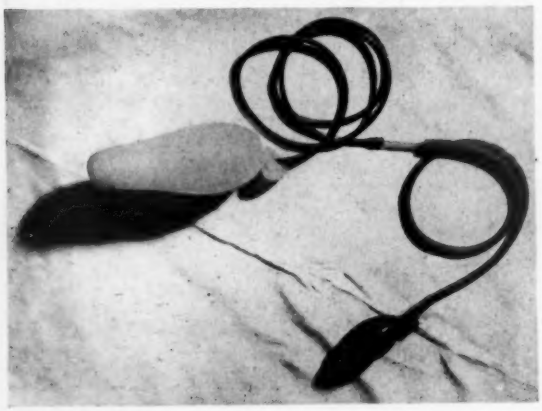

FIG. 1. Apparatus. Showing blood pressure bulb on end of tube with condom tied in place and partly inflated. It must be completely deflated during insertion.

FIG. 2. Roentgenogram of postmortem normal stomach containing a small amount of barium sulfate mixture and markedly inflated balloon.

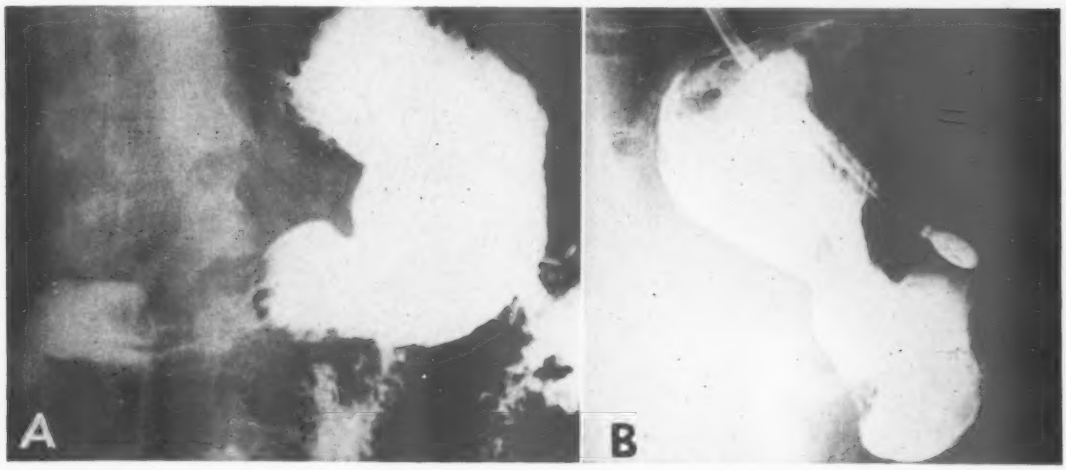

Fig. 3. *A*, usual anteroposterior roentgenogram of normal stomach. *B*, same stomach with inflated balloon. Note gastric outline under diaphragm.

strated very well by their method, but they have no control of the amount of distention.

The best previous discussion of a similar method was given by Gruber[6] in 1945, but he used it primarily from the point of view of physiology. He did include one case of tumor of the stomach, mentioning that the method may have possibilities in diagnosis.

METHOD

Our present method consists of the customary roentgenoscopic examination of the barium-filled stomach with the usual roentgenograms made. The patient then swallows a stomach tube (Rehfuss or Levine) with a latex condom tied tightly proximal to all the holes. A hand bulb from a blood pressure apparatus is attached to the outside end of the tube and under roentgenoscopic control the balloon is in-

Fig. 4. Roentgenogram of normal stomach with balloon taken in right anterior oblique position showing good double contrast effect.

Fig. 5. Normal stomach in anteroposterior position with balloon in the prepyloric area and first part of the duodenum.

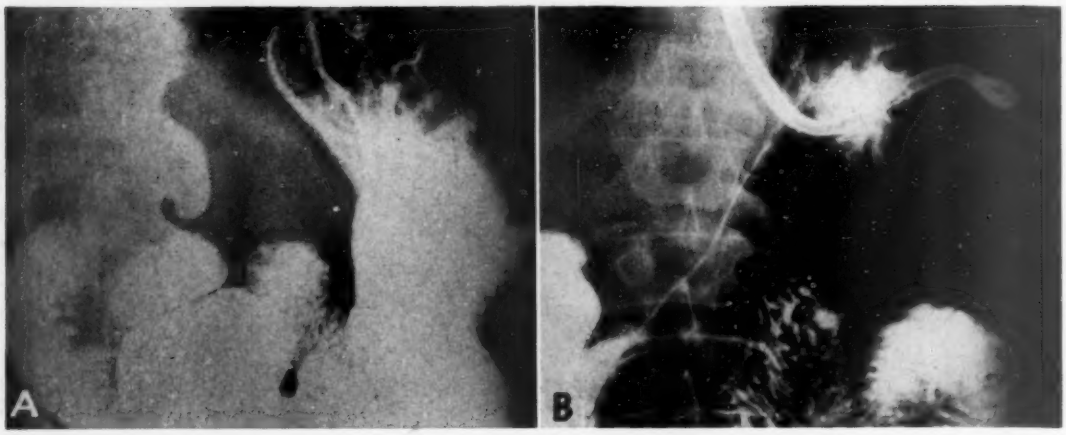

Fig. 6. *A*, usual right anterior oblique position for stomach. Note marked irregularity of mucosal folds of fundus along greater curvature. *B*, double contrast film of same case in same position showing smooth mucosal folds and small hiatus hernia. Gastroscopic examination showed normal stomach.

flated to any desired amount. This, of course, causes a varying degree of gastric dilatation and in most subjects it is possible to palpate the lower end of the balloon in the pyloric antrum, thereby giving transmitted pulsations to the fundus. Tilting of the patient will give control of the amount of barium in the fundus or prepylorus. The maximum pressure used has never exceeded 20 mm. of mercury as measured with a large dial sphygmomanometer.

Fig. 7. Normal stomach showing practically maximum distention. The patient had no complaints referable to the balloon. The intragastric pressure was 20 mm. of mercury.

None of the patients have complained of pain in spite of rather marked distention of the stomach at times. One patient attempted to vomit after inflation of the balloon but nothing happened. Two balloons ruptured while in the stomach but the patient was not aware of what had occurred.

We have been taking spot roentgenograms from time to time but our principal films have been made in the next room. The balloon is left in situ while the patient walks to the roentgenographic machine. Two roentgenograms are taken in the right anterior oblique position which allows the balloon to float into the fundus area and the barium sulfate to descend into the prepylorus. The other two roentgenograms are taken in the left posterior oblique position which gives the opposite effect in that the balloon floats into the prepylorus and the barium enters the fundus. It is best to reduce the peak kilovolts used about eight for films when the balloon is inflated. The roentgenograms are all taken with the patient horizontal.

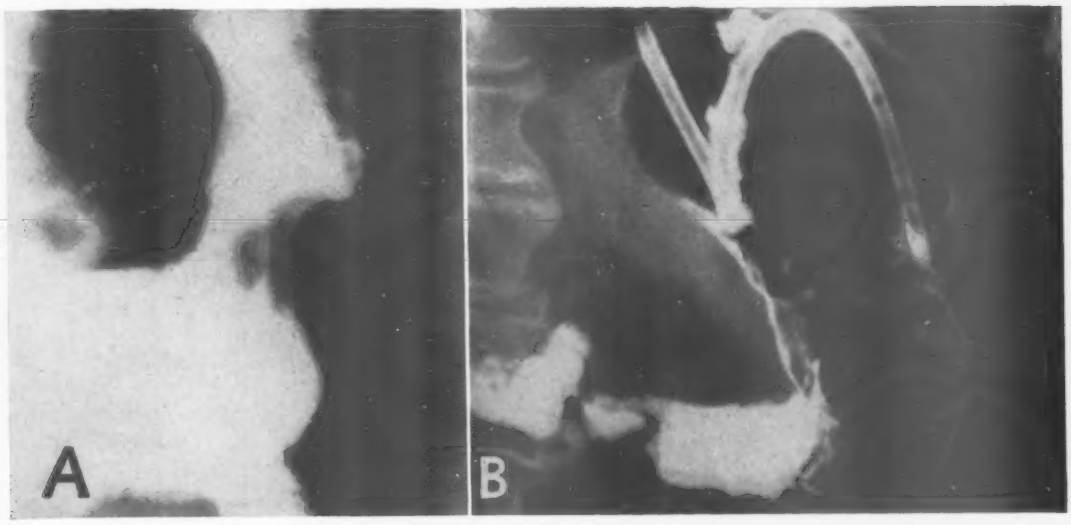

FIG. 8. *A*, spot roentgenogram of stomach showing apparent filling defect that was constant during roentgenoscopy. *B*, same case with double contrast technique showing defect erased by balloon. Patient was later gastroscoped and no pathologic condition found.

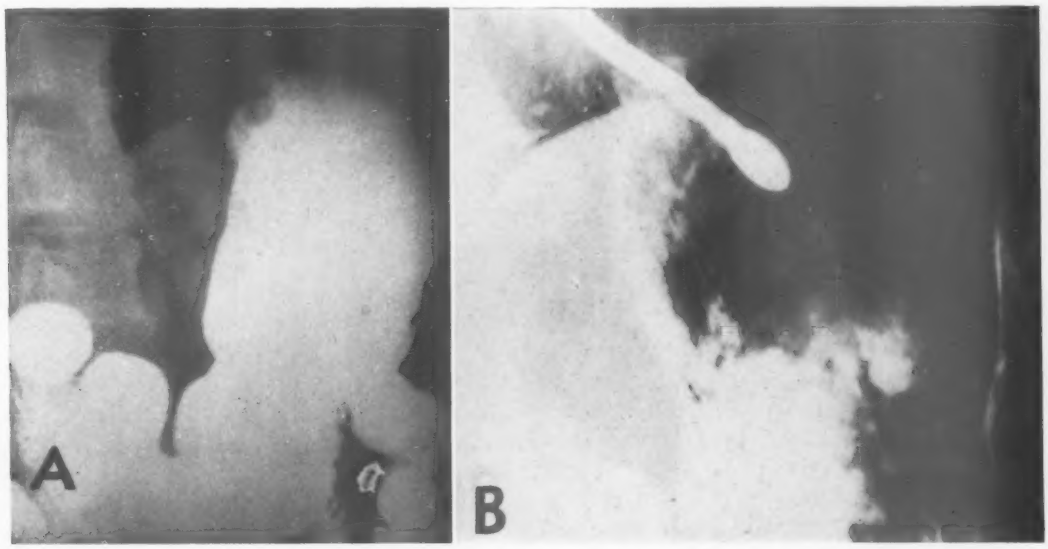

FIG. 9. *A*, usual right anterior oblique film of stomach showing polypoid-like lesion at esophageal opening. *B*, same case with double contrast technique and right anterior oblique position. Film is slightly blurred due to heart motion. There is normal outline seen about the cardia. Patient was explored nevertheless and no pathologic condition found when stomach was opened.

RESULTS

In our experience this method has been a very excellent means of obtaining what amounts to internal palpation of the stomach plus excellent double contrast roentgenograms of almost any area of the stomach. It has been of value in reducing the number of false positive diagnoses, particularly in the fundus where external palpation is not possible. The cases shown in the accompanying illustrations are self-explanatory.

SUMMARY

1. A means of obtaining double contrast roentgenograms of the stomach is presented.

2. This method has been found to be of value particularly in reducing the number of false positive diagnoses.

3. It affords a means of palpation of the fundus of the stomach.

4. The authors feel that this method should be brought to the attention of roentgenologists, not as a panacea for all diagnostic errors but as an additional method in selected puzzling cases.

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THE RELATION OF THERAPEUTIC RADIOLOGY TO CLINICAL MEDICINE*

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GENERAL CONSIDERATION

SINCE the therapeutic radiologist is continually called upon to treat a great variety of lesions, benign, inflammatory and malignant, it is essential that he should have a wide knowledge of the biologic and clinical features of these lesions. The greater is this knowledge, and the more the treatment is based upon it, the more likely are the cases selected for treatment to be chosen wisely, and the more favorable are the results likely to be. No one, I think, would venture to question the importance of this fundamental consideration.

Moreover, the more familiar is the radiologist with the biologic, pathologic and clinical aspects of the conditions which he must treat, the more respect will he command from his colleagues in medicine, pathology, surgery or in other specialties. But a common misfortune is that some radiologists have allowed themselves to lose contact with clinical medicine to such an extent that they can no longer examine patients thoroughly and confidently. As a result, the diagnosis as made by other physicians is accepted without question, and some patients are treated for conditions which they do not have. Under these circumstances, unfavorable results should not surprise anyone.

For this the radiologist may or may not be to blame. When he has not kept abreast of advancing knowledge—and here a sharp distinction must be made between real knowledge and the host of new notions that constitute but a transient medical fashion—he is to blame; when he has allowed his skill in physical examination to deteriorate or when he has failed to develop skill in physical examination, whether this has been because he has worshipped too much at the shrine of the laboratory or because of the common tendency to take things for granted, he is again to blame. For many years the increasing reliance on laboratory procedures has caused a large proportion of physicians, internists, surgeons and others to give less and less attention to physical examination, and radiologists have suffered as much as others from this tendency.

Excessive specialization also has played an increasingly important role in altering the physician's outlook about his work. Specialization has undeniable advantages, but it also has disadvantages which should be recognized more than they are. Without any doubt it tends to increase knowledge in certain directions, but the more one concentrates one's attention to a limited field or to a single phase of a subject, the more this tends to reduce one's mental horizon, and the more one tends to lose one's sense of perspective or fails to develop this sense. Moreover, too early specialization tends to prevent one from acquiring a sense of perspective. The well-known saying that a specialist learns more and more about less and less is entirely true, but the logical conclusion is that, in time, the specialist learns everything about nothing.

During recent years the rapid increase in scientific developments, the tremendous increase in the number of workers in the field of science, and especially the widespread use of publicity for propaganda purposes have resulted in an excessive glorification of science, which is extremely dangerous because it misleads, not only the general public, but some of those who are engaged in scientific pursuits, into be-

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lieving that science can solve not only scientific problems but even political problems. That this is not an exaggeration may be seen by reading Hogben's book, "Dangerous Thoughts."[1] Some seasoned scientists realize this danger and are frankly worried about it. In 1947 Smyth[2] wrote:

> In the past two years science and scientists in this country have achieved a prestige which they have never had before. They have been credited with technical marvels that contributed largely to winning the war, they have lobbied on political questions, they have of necessity talked politics and international affairs. Advertisements have hailed them as harbingers of a new and better—or at least more mechanized—world. More concretely, the federal government is subsidizing basic research in a way hitherto unparalleled. All this is very flattering, but it is also dangerous. The degree of Ph.D. in physics or chemistry or some other science does not automatically stamp a man as great or wise or even very bright. An advanced degree, however, should represent a certain capacity for objectivity; a capacity that I think we might well turn to an examination of ourselves and our science at a time when we may be in danger of arrogance and conceit.

The foregoing factors affect everyone more or less, and physicians, including radiologists, are also affected. The glamour of science and of laboratory procedures in general has a tremendous influence on medical diagnosis, and to a considerable degree on treatment also. The great value of methods of precision cannot be doubted, but when these are applied to biologic material which varies within wide limits, or when they are employed by persons whose skill and experience vary a great deal, the value of the methods diminishes considerably. At the present time, it is fair to say that the majority of physicians rely far too much on the results of laboratory procedures; in fact, many of them base their diagnosis of a patient's illness on the results of this or that laboratory test, and when the tests are not conclusive, they are disappointed and sometimes they berate the clinical pathologists. The inevitable effect of this increasing tendency is that skill in physical examination and the ability to analyze the history of the patient's illness and to correlate it with the physical findings have steadily diminished.

What effect has this had on radiology? As far as diagnosis is concerned, many internists, surgeons and others depend too much on the opinion of the roentgenologist. This may gratify the vanity of the roentgenologist, but it is a dangerous thing because it may easily lead the roentgenologist to assume that he knows much more than he actually does. Because the diagnosis of malignant tumors of bone largely depends on the opinions of roentgenologists, and because many errors result from this excessive and unwarranted reliance, I have not hesitated to make the foregoing comments. Almost as much might be said in relation to tumors of the mediastinum.

For the reasons which I have ventured to mention, the therapeutic radiologist should carefully avoid taking things for granted and accepting without question a diagnosis based largely or wholly on laboratory data, especially when the diagnosis does not explain the patient's major symptoms. He should insist on analyzing the history himself, on examining the patient, and on drawing his own conclusions.

At the beginning I mentioned that the radiologist may or may not be to blame for his failure to acquire a high degree of skill in physical examination, or for his failure to become familiar with the biologic, pathologic and clinical aspects of the great variety of conditions which he may be required to treat, and with the biologic action of the rays. Sometimes he may be only partly responsible for this situation, and in some instances his responsibility is almost nil. The fact is that, in some medical institutions, the attitude of the management or dominance of the work by a short-sighted internist or surgeon may produce an environment in which the therapeutic radiologist may be passively or actively discouraged from doing the best grade of work of which he may be capable.

Usually, when these conditions prevail, the radiologist himself is at least partly to blame, but not always. There is no denying that, in some institutions, personal popularity is more important than knowledge, and this occult factor may be a potent cause of undesirable conditions that may prevent the highest grade of work.

BIOLOGIC CONSIDERATION

Of greatest importance to the therapeutic radiologist is knowledge of the natural sensitiveness of different varieties of normal cells and tissues to roentgen rays, to radium and to other radioactive agents. This knowledge may be acquired by a critical review and analysis of experiments on animals, by analyzing the earlier observations of others in relation to the results of animal experiments, and by personal observation of the action of the rays on patients under treatment for inflammatory or malignant processes. This knowledge is invaluable, not only because it enables one to understand the effects of irradiation, but also because it often enables one, from these effects and from the rate at which they occur, to recognize the character of the lesions. And this may make it possible to verify the diagnosis which has been made or sometimes to show that the diagnosis could not have been accurate. It is unfortunate that only a minority of radiologists have given enough attention to this phase of their specialty to be thoroughly familiar with it and to be able to draw sound conclusions.

The slow and rather characteristic response of tuberculous lesions, the rapid abatement of acute inflammation caused by pyogenic bacteria, and the hastening of suppuration when furuncles or carbuncles are treated with roentgen rays are familiar examples. The gradual improvement of many chronic inflammatory conditions, or of keloid formations, after repeated exposure to suitable doses of roentgen rays is equally illuminating. The rapid and pronounced regression of sensitive neoplasms such as Hodgkin's disease or lymphosarcoma, Wilms' tumors in children, embryonal carcinoma of the testis, endothelial myeloma of bone, or xanthomatosis in children, provides a striking contrast to the much slower response of epithelial tumors, to the comparatively slight and transient regression of neoplasms such as chondrosarcoma or osteogenic sarcoma of bone, and to the complete absence of regression of resistant neoplasms, such as myxosarcomas and hypernephromas.

As the radiologist's knowledge and experience increase, his clinical value increases in proportion; in time his ability to relieve the sufferings of patients or to cure them of malignant conditions with which they may be afflicted commands the respect of his colleagues.

CLINICAL CONSIDERATION

In connection with inflammatory as well as malignant conditions the radiologist should be thoroughly familiar with the clinical and pathologic features of the lesions, and also with the experimental background bearing on the action of the rays. Otherwise, it would be impossible to understand how the lesions are influenced by exposure to the rays. It must be admitted that, in relation to certain morbid conditions, the experimental data as well as the clinical reports do not furnish an absolutely convincing explanation, but often the evidence provides a reasonable or plausible explanation that can be used as a basis for further investigation.

In relation to malignant conditions many physicians know much too little about the natural history of different kinds of tumors; and for a therapeutic radiologist the lack of this knowledge is a serious handicap because, without it, a high grade of work is impossible. Needless to say, the radiologist should know the mode and rate of growth of each kind of neoplasm, the regions or organs most likely to be affected primarily and secondarily, the tendency of each kind of tumor to metastasize, whether metastasis is likely to occur early, late or not at all, and the regions or organs

most likely to become secondarily involved. Knowledge of this kind may be acquired from books, but it should always be carefully correlated with personal observation in as many cases as possible. When a radiologist accepts without question what earlier authors have written, he may be neglecting an excellent opportunity to contribute to the sum total of available knowledge, and he may unconsciously accept information which may be grossly misleading. Too often the writings of others are read without critical analysis, and the ideas and opinions of others are accepted at face value. This is a tacit admission that the reader himself does not know much about the subject and that he does not consider himself competent to judge between contending ideas or doctrines.

PATHOLOGIC CONSIDERATION

As far as verifying or identifying the character of inflammatory or neoplastic conditions is concerned, no one can deny the great value of microscopic examination of sections of tissue by an experienced pathologist. At the present time biopsy has become an invaluable procedure on which most physicians rely. In fact, the question is whether or not physicians rely too much on this procedure and on the opinions of pathologists. While pursuing their studies in school and hospital, most physicians have been so thoroughly indoctrinated concerning the value of biopsy that, throughout the rest of their lives, whenever they receive the opinion of a pathologist on the identity of a tumor, they disregard the history of the patient's illness, its clinical features, and the physical, roentgenologic and other findings; they stop thinking, and they accept the pathologist's opinion without further ado; and this regardless of the fact that sometimes the pathologist's opinion cannot be reconciled with the patient's history, with the clinical features of his illness or with the physical or other findings. What does this mean? It means that the majority of physicians do not know much more about tumors than the meaning of the names by which different kinds of tumors are designated. So great has this trust in pathologists become that, at almost every medical meeting, one or more physicians who read papers have arranged their cases into two groups: "proved" and "unproved" cases. In the group of proved cases are included all those in which tissue from a tumor had been removed and in which sections of the tissue had been examined by a pathologist whose opinion had been accepted forthwith. In the group of unproved cases are included those in which tissue for biopsy had not been removed and in which the opinion of a pathologist had not been sought.

This blind and general reliance on the opinions of pathologists may be gratifying to some of them, but those who realize the tremendous burden of responsibility which is thus placed upon them do not relish it; but what can they do? The fact is that, as far as the identification of tumors is concerned, no pathologist, however experienced he may be, can always be accurate. One of the most eminent pathologists of this generation did not claim an accuracy greater than 85 per cent. It must be evident that, if the ablest pathologists cannot accurately recognize the character of malignant neoplasms in more than 85 per cent of cases, the accuracy of pathologists of lesser caliber is even lower. Many pathologists do not venture to express an opinion on the identity of a tumor without considering the history of a patient's illness as well as its clinical features and the physical findings; but others take a sort of pride in basing their opinion entirely on morphologic cellular changes. The accuracy of the latter is probably even less than that of the former.

Pathologists vary in knowledge and in intelligence as much as other physicians. Those who are teachers often impress their personality on their pupils to such a degree that, throughout their career, these pupils are loath to accept the opinions of other pathologists. Those who work in large hos-

pitals or clinics have a similar influence on other members of the staff. So prevalent is this tendency that, when a patient has been subjected to biopsy at another institution, the physicians of the second hospital refuse to accept the opinion of the pathologist of the first hospital, and either sections are obtained and are re-examined by the pathologist of the second hospital, or the patient is subjected to a second removal of tissue for biopsy. In other words, the attitude of the physicians of each hospital or clinic largely depends on their confidence in the ability of their own pathologist; this attitude, therefore, rests largely on faith.

Different pathologists often cannot agree on the essential character of a neoplasm. Disagreements of this kind are much more common than most physicians realize. Some pathologists, who have made a special study of epithelial tumors, often find "epithelioma" or "carcinoma" when other pathologists think otherwise or when the history and the physical findings indicate some other kind of neoplastic process. Other pathologists, who have given special attention to some other variety of tumor, tend to find evidence of this tumor when others to whom the same sections may be submitted find something else. When the same sections are sent to six different pathologists, it is not rare to obtain two, three or more different opinions. It must be apparent, therefore, that different pathologists have different criteria.

There is no doubt that microscopic examination of sections of a tumor by an experienced pathologist is the best single method of identifying a neoplasm that is available at the present time. But from the foregoing considerations it is evident that blind reliance on the opinions of the pathologists is not altogether warranted. When the opinion of a pathologist cannot be reconciled with the history, with the clinical features of the disease, or with the physical or roentgenologic findings, it should be received with the utmost skepticism. Sometimes, also, the biologic response (rate and degree of regression) of a tumor to roentgen irradiation can furnish an invaluable clue to its character, and sometimes the evidence thus obtained is even more important than microscopic inspection of sections by a pathologist. But the best method of all is a critical analysis of all these elements.

PHYSICS AND RADIOLOGY

If a well-qualified therapeutic radiologist from another country were to come to the United States in order to see what American radiologists are up to and how they conduct their work, what would be his impressions? Perhaps his strongest impression, as far as the treatment of malignant neoplasms is concerned, would be that the majority of American radiologists treat all varieties of tumors essentially by the same method; and this regardless of the relative sensitiveness or resistance of different kinds of neoplasms. By this I mean that the majority of malignant processes are treated with roentgen rays generated at 200, 400, 600, 1,000 or even 2,000 kilovolts, and that the treatment is given by some variation of the fractional method. The total surface and tumor dose may be given in ten, fifteen, twenty, thirty or forty days, or even longer, and this dose may be given through one, two, three, four or more fields. But much the same method is employed whether the malignant condition is exceptionally sensitive to irradiation, as in Hodgkin's disease or lymphosarcoma, whether the tumor is only moderately sensitive, as in epithelioma or carcinoma, or whether it has a high degree of resistance, as in hypernephroma, mixed tumor of the parotid gland or myxosarcoma. This rather general tendency is not confined to this country; many foreign radiologists have tended to follow the same course.

How and why has this general uniformity in method of treatment developed? Because of several factors, among which perhaps the most important have been: (1) the desire to "standardize" treatment as much as possible; (2) the increasing de-

mand for rays of greater penetration; (3) the fact that, other things being equal, rays of different wavelengths have the same effect on cells and tissues; (4) the natural desire to avoid undesirable effects in the skin, and (5) the desire to diminish radiation sickness as much as possible.

To me, at least, it would seem that standardization of treatment has gone too far. There is no doubt that the other factors mentioned are valid and desirable up to a certain point, but not to the point of sacrificing still more important fundamental biologic considerations. The reason why the foregoing factors have been so generally adopted is not difficult to understand. For a number of years, at the annual meetings of the radiologic societies, courses of instruction have been held, and these have been deservedly popular. Among these courses one of the most popular has been the course on radiation physics, usually given by an outstanding physicist.

Given a homogeneous substance in a mass of definite size, a physicist can readily furnish exact figures on its density, specific gravity, permeability to water, heat and so forth. But when the substance is not homogeneous or when the size of the mass is irregular, the physicist no longer can furnish exact figures. For him the problem becomes increasingly difficult when the substance whose properties are to be measured is a mass of living tissues and cells which vary greatly in their composition, physiology and metabolism. When a physicist undertakes to measure the penetration and absorption of roentgen rays of a given quality in a substance of uniform composition, such as water, gelatin, wood or steel of a given density, the problem is relatively simple. He can even measure with accuracy the penetration and absorption of rays of a given quality in living tissue, but these measurements, however accurate they may be, do not give any indication of the relative natural sensitiveness or resistance of the tissue; they cannot take into account the different composition of different parts of the tissue, its blood and nerve supply, the great differences in metabolism of different kinds of cells under different conditions, or the biologic changes that occur after exposure to different substances or agents.

If this is true of normal cells and tissues, it is just as true of malignant tumors. To a physicist a neoplasm is a mass of tissue; to him the differences in the natural history of different kinds of tumors, their tendency to metastasize early or late, the tissues or organs which are prone to be affected primarily or secondarily, and their great difference in sensitiveness or resistance to roentgen rays or to radium, mean very little. But to a therapeutic radiologist these biologic considerations should be at least as important as the penetration of the rays or as the number of roentgens that can be delivered to a certain depth in a certain number of days.

To illustrate what I mean, perhaps I may be allowed to use a pertinent example. The fractional method of irradiation or, as it is often called, the Coutard method, was based on the results of animal experiments; these had shown that, when a small dose of roentgen rays is given to a certain area every day, this can be continued until a very large total dose has been attained. When the total dose is sufficient, and when it is given within a certain number of days, it produces in the irradiated skin or mucous membrane a reaction the degree of which varies with the dose. When a small number of roentgens is given daily, or when the total dose is given over a large number of days, a much larger total dose can be given without permanent injury to the skin or other tissues. Other experiments having shown that, in order to arrest permanently the growth of a malignant epithelial tumor, from five to eight times an erythema dose is required, the fractional method of treatment is the only way in which a cancerocidal dose can be given without risk of serious and permanent injury.

From the foregoing considerations it is apparent that the fractional method of irradiation was designed to deal with

epithelial neoplasms or with other neoplasms that have an equal degree of sensitiveness to roentgen rays or that are even more resistant to the rays than epitheliomas or carcinomas. But instead of limiting the use of this method to epithelial and other resistant tumors, the majority of radiologists employ the same method to treat all malignant neoplasms, even the exceptionally sensitive tumors that arise in lymphoid structures, such as in Hodgkin's disease or lymphosarcoma. Some radiologists, it is true, do not give as large total doses as in the case of epithelial neoplasms, but others treat all tumors by the same method and with approximately the same total dose.

For several reasons this is an unfortunate practice which can be accounted for only by unfamiliarity with many of the clinical aspects of the different forms of lymphoblastoma, by lack of experience with a sufficient number of cases, and by the teachings of physicists who do not realize the great difference between epithelial and lymphoid tumors. Physicians in general have been taught, and they believe, that Hodgkin's disease or lymphosarcoma nearly always begins in the neck and spreads from the cervical nodes to the axillary and inguinal nodes. Most of the earlier writers believed this to be true, and some recent writers, such as Jackson and Parker,[3] have stated that Hodgkin's disease nearly always begins in the neck. It is undoubtedly for this reason that, when therapeutic radiologists are called upon to treat a lesion of this kind, most of them limit their treatment to the neck, armpits and groins, although sometimes the mediastinum also may be treated.

The truth is that Hodgkin's disease begins in the cervical lymph nodes or in the nasopharynx or tonsil in about half the cases, or even less often. In an equal or even greater number of cases this condition begins in the retro-abdominal (para-aortic, mesenteric or iliac) lymph nodes. It begins in the mediastinal nodes only in a small proportion of cases; usually when these nodes are affected, their involvement is secondary to earlier involvement of the cervical or retroperitoneal nodes. Only rarely does it begin in the axillary or inguinal nodes; nearly always, when the axillary nodes are affected, some of the mediastinal nodes were primarily or secondarily affected long before. And nearly always, when the inguinal nodes are involved, the retro-abdominal nodes have been involved for a long time. Lymphosarcoma may begin in any group of lymph nodes or in any lymphoid structure in the body, but once the malignant process has started, it tends to spread to other groups of lymph nodes in the same manner as Hodgkin's disease. This is also true of other forms of lymphoblastoma. Unfortunately, these important clinical features are not generally known, and this is one reason why many radiologists do not treat these diseases as well as they might be treated and why the results often are not as good as they might be.

But aside from these clinical considerations, the morbid process which, in these conditions, affects the lymph nodes and other lymphoid structures is so exceptionally sensitive to roentgen rays that the fractional method of irradiation, especially with rays generated at 200 or more kilovolts, does not yield as good results, immediately and for the long run, as another method which I have described in another paper.[4] Instead of giving a small daily fractional dose for many successive days until a maximal total dose has been attained, each of the fields requiring treatment is given, in a single session on one day, a suberythema dose (550 r) or, if the patient cannot tolerate such a dose, each field receives half of this dose each day on two successive days. Naturally the number of fields requiring treatment varies in different patients at any given time according to the groups of nodes in different regions that are involved and that are responsible for the patient's symptoms.

Only a small number of patients who are afflicted with Hodgkin's disease or lympho-

sarcoma are first seen by a physician when only a few cervical nodes are involved and when lymph nodes in other parts of the body are not affected. Then it is sometimes possible, by thorough treatment, to bring about a complete and permanent cure. This can be done in one of two ways: (1) by giving to the affected region a course of fractional irradiation with a total dose of 2,000 to 3,000 r (rays generated at any voltage between 130 and 200 kv., or more), or (2) by giving to the involved area a dose of 550 r of rays generated at 130 or 140 kv., and by repeating this twice at intervals of three weeks.

In the great majority of cases, however, the malignant process, when the patients first consult a physician, has already invaded more than one group of lymph nodes, and sometimes the condition affects several groups of nodes, not to mention the spleen or the liver. Under these circumstances, any hope of a complete and permanent cure must be abandoned; but the patient's condition may be greatly improved by suitable treatment, and not infrequently this improvement can be maintained for many months or even many years. Radiologists who employ the fractional method with rays generated at 200 kv. or more may be gratified by the immediate effect of treatment on certain regions, but later, when the morbid condition again becomes active and when additional treatment is given in the same manner, the response is less favorable than it was the first time. After treatment by the fractional method has been repeated two or three times, it usually ceases to have any favorable effect, treatment with roentgen rays must be abandoned, and the patient no longer can look forward to even temporary improvement when the former symptoms recur or when fresh symptoms develop.

But this is not true when the fractional method has not been employed, when the fields requiring treatment have received a suberythema dose (in one or two days) of rays generated at 130 or 140 kv., when treatment has been repeated once or twice at intervals of three or four weeks. When the treatment is arranged in this manner, it usually continues to have a favorable effect for many months or years, until the malignant condition reaches an advanced stage or until it enters the terminal phase.

I have used these two examples because they illustrate so strikingly the importance, for the therapeutic radiologist, of knowledge of the natural history of different kinds of tumors, of their clinical features, and also of avoiding too much standardization in treating them. Other examples could be described, but space forbids.

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DISCUSSION

DR. JAMES T. CASE, Chicago, Ill. The remarks we have just heard constitute an able presentation of a most important theme which should never be lost sight of by radiologists, whether their interest is devoted more to therapy or to diagnosis.

A famous surgeon once remarked that he was a physician practicing surgery. We are physicians practicing radiology. Too often, some of our colleagues unintentionally (but thus revealing their innermost thought) refer to us as "x-ray men." This is largely the fault of those radiologists who neglect their opportunities to participate in the phase of medical practice which is usually referred to as clinical, as contrasted with the radiological aspect.

The dictionary definition of "clinical medicine" says that it is that part of medicine or surgery dealing with the investigation of disease. in the living subject. It is certainly incorrect to build up a contrast between radiological and

clinical findings. The roentgen examination is a very important part of a clinical examination. Indeed, there are few maladies in which the roentgenologic study does not play some part in the clinical examination or in treatment.

We have had presented at this meeting numerous examples of the collaboration necessary in the study of the chest, for example. Certainly no persuasion is needed to accept the theme of these remarks. Since radiology does enter so widely into the field of medicine and surgery in general as well as many of the specialties, it means that of all groups of physicians, the radiological group will be going to school for the rest of their lives in order to keep abreast of the advances in general medicine and the new developments in methods of examination apart from our own specialty.

PYELOGRAPHY BY RAPID INJECTION*

DESCRIPTION OF AN IMPROVED METHOD AND RESULTS IN 200 CASES

By LOUIS J. FRIEDMAN, HERBERT LOBSENZ *and* RUSSELL JAUERNIG

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INTRAVENOUS pyelography, the method of forming a roentgenological shadow of the kidney and its appendages by the intravenous injection of a radiopaque compound which is eliminated in high concentration in the urine, is by now so firmly established as an informative and safe procedure that we shall offer no discussion of its merits. Present-day experience centers about two such compounds or media:

(1) 3,5-diiodo-4-pyridone-N-acetic acid, prepared in a solution of diethanolamine, furnished commercially as "diodrast";

(2) disodium N-methyl-3,5-diiodo-chelidamate, an aqueous solution furnished commercially as "neo-iopax."

Both media are accepted by the Council on Pharmacy and Chemistry; both produce excellent, workable views of the urinary tract following intravenous injection.

Neo-iopax, while perhaps not responsible for even a single death,[2,4,5,11] has the disadvantage of occasionally producing acute pain in the injected arm during and for several minutes following administration.

The subject of the present report is a method of injecting neo-iopax that almost entirely eliminates the characteristic arm pain. It will show that rapid intravenous injection is the method of choice. Constitutional reactions to the injection were evaluated in an uncontrolled series of cases.

Instructions provided by the manufacturer of neo-iopax with each package state: "The contents are then injected slowly into the cubital vein." We disagree with this recommendation.

The same instructions are in error on the side of caution in a second respect: under "Contraindications" they state, "The use of the drug is contraindicated in patients with severe liver disorders, nephritis or hyperthyroidism, and great care must be exercised in cases of uremia. Preliminary liver and kidney function tests are advisable in suspected cases. Caution must also be exercised in patients with any severe systematic disease." We have disregarded these contraindications.[3,12] They were evidently suggested by the consultative staff of the manufacturer on the ground of any *possible* derangements in physiology which might be adversely affected and are not conclusions from actual untoward results. From our own experience these do not appear significant. We feel that this is important, since urographic information is often desirable in precisely the conditions named, and especially during established renal insufficiency the cause of which is still uncertain.

A third observation is that true sensitivity reactions[13] did not appear in any case in our series. The recommended tests for sensitivity are (1) injection of a small amount, such as 1 or 2 cc., followed by a waiting period, and (2) the sublingual application of 1 cc. of solution, followed by examination of the mucosa in ten minutes for signs of edema, and after thirty minutes (the medium having been swallowed) general survey of the patient for allergic responses. We have simplified the test as follows: An injection of 0.1 cc. is made intravenously and the syringe left in situ. If no evidence of systemic reaction (by which we do not mean such incidental mild reactions as flushing or warmth) appears within thirty seconds the injection is completed. In all cases, regardless of the patient's weight, with the exception of small

* From the Roentgenological Service of the X-ray Division, Bellevue Hospital, New York.

children,[6] we used 20 cc. of 50 per cent neoiopax.

The cases presented are 200 routine, successive, unselected patients undergoing urography in this department at the request of the various services under whose care they were. (Actually only 199 *different* patients are included, one having been examined twice during the series: entries 99 and 181.) Contraindications were disregarded, although it is evident from the diagnoses that the contraindications mentioned occurred frequently. The injection was made rapidly (in less than one minute) in practically all cases. To allow a free rapid venous return, the forearm was extended and the arm abducted to a position perpendicular to the body. The films thus produced demonstrated shadows at least equal to those obtained in our experience with this and other media in similar cases using the so-called standard routine.[1,7,8,10,14,15,16]

Table 1 lists all our cases seriatim, with race, sex, age, diagnosis and reactions, if any. The usual entries are self explanatory, as to race (w white, c colored, y yellow), sex (m, f) and age. Since the diagnosis in many patients was not yet established at the time of the examination, as this was indeed the purpose of the urography, it often happened that our information was limited to the leading signs or symptoms; in that event we have indicated whatever information was available (using parentheses):

? Renal pathology (edema, albuminuria, casts, low spec. grav.)
(Pain in LUQ)
Burns, 3rd degree, of axilla (albuminuria; pyuria)

The customary abbreviations have been employed where convenient:

RUQ right upper quadrant (of the abdomen)
LLQ left lower quadrant
c.v.a. costovertebral angle
C.V.D. cardiovascular disease
C.N.S. central nervous system

Allusions to right and left have been largely omitted from the table, except when material, as:

(Pain, right c.v.a. Nephrectomy 1924 reason unknown, left)

In describing the reactions attributable to the urographic medium, we have found it useful to abbreviate, using p for pain and b for burning, if slight in degree; *severe* pain or burning by P or B. Unless otherwise described, these reactions were limited to the injection site. In addition, we have designated infiltration by i, and erythema by e. General reactions have been abbreviated as follows:

b burning (other than locally)
d dyspnea
e (emesis) vomiting
f flushing (the site mentioned)
h headache
n nausea
r retching
s sweating (the site mentioned)
v (vertigo) dizziness
w sensation of warmth

In evaluating the distress caused by any injection, it is important to distinguish between expressions of "pain" and of "burning," and to discount such occurrences as "slight nausea" and "dizziness," since low grade complaints are common to nervous patients receiving an injection of anything whatever, even of sterile water; Case 160 is an interesting example of a purely emotional responses which might have passed for an important constitutional reaction, viz., a shaking chill, which could be stopped on command.

In the case of infiltration, a mishap that can never be wholly prevented and that is peculiarly troublesome when working with the small veins of children, some localized pain must be expected, whether the material be given rapidly or slowly; we do not, therefore, count these accidents as a feature of the technique under study. In one case (a child) the material was injected deep intramuscularly and no severe pain was noted. No absorption of medium was demonstrable over a period of three hours.

TABLE I

	Case	Sex	Age	Diagnosis	Local Reaction	General Reaction	Remarks
1	G.S.	w f	29	Pyelonephritis, acute bilat.	none	n 10 min. after inj.	
2	D.G.	w m	28	Paraplegia; cystitis	i, P	none	controlled by 1% novocaine
3	C.S.	f	4	? Renal pathology (recurrent frequency and fever)	none	none	dose 15 cc.
4	J.V.	f	25	? Renal tuberculosis	none	w during, n after inj.	
5	L.M.	f	40	? Renal tuberculosis	none	none	
6	F.H.	f	34	? Renal mass. Cirrhosis of liver	b during inj.	none	disappeared immed. after inj.
7	F.L.	f	32	Pyelonephritis, chronic	b, p	none	
8	J.M.	f	31	(Frequency, urgency, RUQ pain)	none	none	
9	J.S.	m	64	(Urinary retention)	b	none	
10	V.D.P.	m	64	(Renal colic)	none	none	
11	J.M.	f	31	Pyelitis	none	none	
12	W.B.	m	71	. . . (deferred)	none	none	
13	M.D.P.	m	71	Renal lithiasis	none	none	
14	T.B.	m	66	Possible renal disease	i, b	s lower extremities	
15	C.V.	f	42	(Occasional back pain and suprapubic burning)	i, b	none	
16	E.L.	f	42	Essential hypertension	none	v	
17	C.A.	f	54	Pyelonephritis, chronic	none	w, f, v	
18	A.R.	m	72	Obstructive jaundice	none	none	
19	J.M.	m	59	. . .	none	w	
20	M.B.	f	54	(Pain in left flank)	none	v	
21	M.W.	f	54	(Back pain, frequency)	none	none	
22	A.D.	f	32	Renal calculi	none	none	
23	A.I.	f	57	Carcinoma of cervix; ? renal pathology	none	"cold mouth"	
24	H.M.	m	5	Hydronephrosis	ic b	none	dose 12 cc.
25	H.R.	m	37	Pelvic abscess; appendectomy	b	none	
26	M.S.	c f	54	Renal disease undetermined	none	none	
27	R.V.W.	m	43	(Cramping RUQ pain radiating to right c.v.a., acute onset)	none	none	
28	H.M.	m	5	Hydronephrosis	i, b	none	dose 12 cc.
29	L.P.	f	55	Paralysis agitans	b	none	
30	A.A.	f	66	Carcinoma of uterus	none	none	dose 5 cc.
31	M.B.	f	75	Hypertensive C.V.D. (decomp.)	b	n	
32	L.K.	f	43	Syphilis of C.N.S.	none	none	
33	A.McM.	f	30	(c.v.a. pain, chills, fever)	p shoulder	n transitory	
34	M.M.	f	28	? Renal calculus	none	none	
35	H.Z.	f	46	Carcinoma of cervix	none	none	
36	W.M.	f	53	Multiple sclerosis	p to shoulder	none	
37	L.C.	w f	57	Arteriosclerosive and hypertensive heart disease	none	none	
38	M.O.	c f	29	Pyelitis? Renal calculus?	p in shoulder	none	
39	E.M.	w f	65	. . . (deferred)	none	none	
40	N.McS.	w m	36	(Low back pain, 3 years)	p in shoulder	burning in mouth	
41	I.K.	w m	45	Fever of unknown origin	none	none	
42	E.McH.	w f	30	. . .	none	none	
43	C.C.	w f	1	Pyelitis, recurrent	none	none	20 cc. intramuscularly
44	L.S.	w f	38	(Lumbar pain, burning dysuria)	none	none	
45	A.W.	w m	65	Chronic nephritis	none	none	
46	A.M.	w m	57	(Pain L flank, increasing urinary frequency)	none	none	
47	C.S.	c f	68	Carcinoma of rectum (and vaginal bleeding)	b	none	
48	O.C.	c m	22	? Essential hypertension	none	n, v	unreliable?
49	A.L.	w f	34	Pulmonary tuberculosis	none	v	
50	D.G.	c f	56	Hypertensive C.V.D. and gastroenteritis	p shoulder	none	
51	H.C.	c f	24	Pulmonary tuberculosis	none	none	
52	J.R.	w m	56	Fistula in ano	none	n transitory	
53	J.T.	w m	75	Benign prostatic hypertrophy	none	none	
54	W.M.	w m	71	Benign prostatic hypertrophy with urinary retention	none	none	
55	E.L.	w m	17	Possible pyelonephritis	none	none	
56	Y.L.	w f	52	Hematuria, cause unknown	b	none	
57	P.K.	w m	62	Carcinoma of prostate	i, b	none	
58	M.S.	w m	65	Carcinoma of rectum	none	none	

TABLE I—(*continued*)

	Case	Sex	Age	Diagnosis	Local Reaction	General Reaction	Remarks
59	C.J.	c f	32	Pulmonary tuberculosis	none	none	
60	H.S.	c m	35	Pulmonary (? renal) tuberculosis	swelling	none	
61	L.V.	w f	31	Carcinoma of fundus of uterus	e	n	
62	H.M.	c m	37	? Pulmonary tuberculosis	none	e sudden	within 1 min.
63	H.S.	w m	66	Benign prostatic hypertrophy	p shoulder	v, n, thirst	
64	J.M.	w m	41	(Weakness; puffiness of face)	p shoulder	none	
65	R.L.	w f	53	(Back pain. Asthma 8 yr.)	none	none	
66	MB	w f	48	Hypertensive C.V.D. (blindness, headaches, weight loss)	none	none	
67	D.W.	w f	77	? Carcinoma, site unknown (Weight loss, anemia, ? mass in abdomen). Hypertension (hematuria)	none	none	
68	M.G.	c f	59	Nephrectomy 1940 (? traumatic)	none	none	
69	M.Y.	w m	57	Cirrhosis of liver (ascites, jaundice)	none	none	
70	A.McA.	w f	36	Cholecystectomy: postoperative. (Painless hematuria, 3 days)	none	none	
71	S.M.	w f	45	(Pain, right c.v.a. Nephrectomy 1924 reason unknown. L)	none	none	
72	J.W.	w f	48	(Slight hematuria)	none	none	
73	M.T.	w f	52	Carcinoma of cervix	none	none	
74	T.K.	w f	49	Hypertension. (Flank pain, radiating downward; frequency)	none	none	
75	B.F.	c m	69	Carcinoma of bladder	none	none	
76	E.Z.	PR m		Schistosomiasis of bladder (hematuria)	none	v	
77	P.O'B.	w m	13	? Glomerulonephritis ? Calculus	none	v, n	
78	TR	w f	33	Carcinoma of cervix	none	v, n	
79	R.D.	w f	46	(Hematuria)	none	none	
80	A.C.	w m	68	Glomerulonephritis, nephrotic	none	none	
81	E.M.	w f	57	Possible stricture of ureter	none	none	
82	R.S.	c f	28	Post-nephrectomy discharging sinus (Weight loss, ankle and facial edema)	none	none	
83	K.S.	w m	67	Ureterocutaneous fistula Ureteritis cystica	"pins and needles"	none	
84	L.D.	w f	25	Cushing's syndrome	none	none	
85	V.L.	w f	40	Staghorn calculus	none	e after 1 min.	very nervous prior to inj.
86	B.T.	c f	19	Chorionepithelioma	none	none	
87	S.G.	w f	57	(Painful mass LLQ)	b	none	
88	M.B.	w m	77	Post-suprapubic prostatectomy	none	none	
89	JG	c f	27	Lymphadenopathy	none	none	
90	C.C.	w f	21	Pulmonary tuberculosis. Epilepsy. ? Renal tuberculosis	none	none	
91	H.W.	w m		Renal calculus	none	none	
92	S.W.	w m	46	Flank pain, severe, radiating to groin: Renal calculus to be ruled out	i, b	none	
93	I.S.	w m	57	Leukemia	none	none	
94	J.B.	c m	38	Pulmonary tuberculosis. ? Renal tbc	none	none	
95	T.T.	w f	54	Malignant hypertension	none	none	
96	P.S.	w m	42	Pulmonary tuberculosis. ? Renal tbc	none	none	
97	J.G.	w m	47	? Renal calculus	none	none	
98	MN	w f	52	(Mass RUQ = ? Kidney)	none	none	
99	E.K.	w f	58	Carcinoma of cervix	none	none	
100	H.P.	w m	57	Urethral stricture, old. (Pyuria)	none	none	
100	A.M.D.	c f	21	Gestation, intra-uterine	none	none	
102	B.W.	w f	62	(Pain in flank)	none	none	
103	A.McM.	w f	30	Stricture of ureter	none	none	
104	M.M.	w f	60	Papilloma of bladder	none	n	
105	M.A.	w f	7	Renal anomalies	none	none	dose 10 cc.
106	J.O.	w m	55	Typhoid carrier	none	none	
107	R.B.	w f	37	. . . (Pain in c.v.a.)	b	none	
108	J.M.	w f	44	Retrogastric mass	none	none	
109	V.A.	w f	59	Pyelitis	none	n, h	
110	E.B.	w f	40	Hypertension	none	none	
111	M.D.	w f		Tuberculous peritonitis	none	p 1 min. below L costal margin	
112	C.B.	w m	61	Uremia	none	none	
113	J.J.N.	w m	54	Abdominal neoplasm	none	none	
114	E.Z.	w m	57	Pneumoconiosis. Lung abscess	b	none	
115	J.J.	c m	54	. . . (Radiating mid-back pain)	none	none	
116	W.G.	c m	42	Hypertension	none	none	

TABLE I—(*continued*)

	Case	Sex	Age	Diagnosis	Local Reaction	General Reaction	Remarks
117	L.N.	w f	42	Renal lithiasis	none	b mouth and pubic area f face	
118	J.L.	w m	59	Renal calculus	p along arm above site of injection	none	
119	T.F.	w m	45	? Renal calculus (lumbar pain)	none	"menthol" taste in mouth	
120	J.M.	w m	61	Carcinoma of kidney	none	n, r, momentary d	
121	B.T.	c f	30	Carcinoma of cervix	none	none	
122	M.G.	w f	72	Hypertensive C.V.D. (enlarged heart)	none	h	
123	G.K.	w m	20	Pyelonephritis	none	none	
124	Y.C.A.	y m	28	Possible renal calculus	none	none	
125	R.G.	w f	27	? Renal disease	none	v, n	
126	D.S.	w m	68	Carcinoma, ? site (enlarged L cervical nodes)	none	none	
127	F.T.	w f	64	(Abdominal mass)	none	none	
128	J.F.	w m	44	Hypertension, severe	none	none	
129	A.B.	w f	29	Transverse myelitis. Spastic paraplegia	p, b along arm	none	relieved on flexion of elbow
130	P.B.	w m	44	Funiculitis	none	none	
131	M.W.	w f	48	? Renal calculi (Pain in flank)	p mild aching	"chilly feeling" in abdomen	unreliable person
132	J.C.	w m	50	. . .	none	n, r, b	
133	D.B.	w m	64	? Renal calculus	none	none	
134	J.S.	w m		Chronic nephritis. ? Polycystic disease	none	none	
135	B.G.	w f	35	Ureteral stricture	none	none	
136	M.W.	w f	80	(Pain in flank, persistent)	none	none	
137	J.L.	w m	25	? Renal pathology (Back pain, pyuria)	none	none	
138	P.O'B.	w m	13	? Bifid renal pelvis, bilateral (Hematuria, pain on urination)	none	none	
139	G.A.	w m	53	Burns, 3rd degree, of axilla (Albuminuria; pyuria)	none	none	
140	A.H.	c f	44	Post-nephrolithotomy for calculus	none	none	
141	T.V.	w m		Hypertension	none	none	
142	A.R.	w f	53	Hypertensive C.V.D.	none	none	
143	N.E.	w f		Renal tuberculosis	i, p, e	p renal areas	i 1 cc. after 12 cc. had been injected
144	M.H.	w f	36	Hypertensive C.V.D. with uremia	none	none	r, e before inj.
145	E.K.	w f	58	Hypertensive C.V.D.	none	none	
146	H.H.	w m	31	(Abdominal pain, fever)	none	none	
147	L.D.	w m	39	Fractured pelvis. Ruptured bladder	none	none	
148	C.S.	w f	43	Post-nephrotomy for calculi	none	none	
149	S.B.	w m	64	Reticulum-cell sarcoma of spleen and lymph nodes	none	none	
150	F.P.	w f	47	(Pain in LUQ)	none	none	
151	G.V.L.	w m	68	Congestive failure, 6 mo. (Hematuria)	none	none	
152	A.C.	w f	43	? Renal pathology (Edema, albuminuria, casts, low spec. grav.)	none	none	
153	E.P.	w f	47	Carcinoma of cervix 1942. ? Radiation cystitis	none	none	
154	I.C.	c f	58	? Pyelonephritis	none	w throat and perineum; v	
155	R.K.	w m	38	(Mass in LUQ. Anemia)	none	none	
156	J.Z.	w m	59	(Mass in LUQ)	none	none	
157	M.W.	c f	34	Post-hysterectomy: ? ureteral obstruction	none	n; b in operated area	
158	F.L.	w f	86	Pyelonephritis	none	none	
159	T.B.	w m	40	Pulmonary tuberculosis	none	none	
160	F.T.	w f	60	Ureteral calculus	none	shaking chill after 2 min. (emotional)	chill stopped when ordered to remain quiet for film
161	V.M.	w f	4	Pyelonephritis	none	none	dose 10 cc.
162	M.M.	w f	53	Hypertension	none	none	
163	J.C.	w m	50	Probable conversion pain	none	none	
164	S.K.	w m	18	Renal calculus	none	e	
165	A.H.	w f	53	Carcinoma of cervix	none	none	

TABLE I—(*continued*)

	Case	Sex	Age	Diagnosis	Local Reaction	General Reaction	Remarks
166	G.S.	w f	56	Carcinoma of cervix	none	none	
167	L.C.	w m	72	Pyelonephritis	none	none	
168	M.E.	w f	48	? Hydronephrosis	none	none	
169	L.H.	w f	28	Hypertensive C.V.D. Pyelonephritis	none	none	
170	H.B.	w m	75	Carcinoma of prostate. Metastases	none	none	
171	S.Y.P.	y m	88	? Renal neoplasm (Hematuria)	none	none	
172	M.S.	w m	82	Pyelonephritis	none	none	
173	K.C.	w f	46	Carcinoma of cervix	none	none	
174	N.S.	w f		. . . (Deferred)	none	none	
175	J.D.	w m	47	Renal calculus	none	none	
176	E.W.	w f	13	Osteomyelitis of tibia	none	n	dose 18 cc.
177	Y.Y.	y m	53	(Pain and rigidity LUQ)	none	none	
178	W.R.	w m	62	? Renal calculus	none	none	
179	F.B.	w f	63	(Pain in flank, 10 yr.)	none	none	
180	V.F.	w m	59	Cystitis	none	"numbness" in abdomen	
181	E.K.	w f	58	Carcinoma of cervix	none	none	same as Case 99
182	A.H.	c m	54	Hypertensive and arteriosclerotic heart disease	none	none	
183	J.K.	w m	37	? Polycystic kidneys	none	none	
184	S.S.	w m	72	? Carcinoma of kidney	none	none	
185	A.C.	c m		Rheumatic fever, acute. Rheumatic heart disease	none	none	given over a 3 min. period
186	W.K.	c m	52	Hypertensive cardiovascular disease. Nephrosclerosis	none	none	
187	N.C.	w m	73	Carcinoma of prostate	none	none	
188	D.J.	w f	53	Urolithiasis	none	none	
189	A.C.	w f	32	? Renal neoplasm (abdominal mass and pleural effusion)	none	none	
190	A.K.	w f	28	(Pain, suprapubic and in flank)	aching in shoulder and arm for 4 min.	n, r for 2 min., chills and general trembling for 4 min.	20 cc. given slowly in 2–3 cc. portions in 5 min. History of hay fever and e after diodrast
191	S.B.	w f	45	(Pain in flank, nocturia, frequency)	none	none	
192	L.D.	w f	27	? Ureteral calculus	none	n	
193	A.J.	w f	28	Carcinoma of cervix	none	none	
194	R.M.	w f	19	Congenital heart dis. Epilepsy (Nausea and vomiting)	none	n	
195	L.M.	w f	43	Pyelonephritis, acute	none	none	
196	S.K.	w m	64	Cholecystitis. Hypertension	none	none	
197	J.C.	w m	7	Congential obstruction causing retention	none	none	dose 10 cc.
198	L.G.	w m	35	(Pain over both kidney areas)	none	n	
199	A.S.	w m	70	Chronic nephritis	none	none	
200	M.F.	w f	69	(Acute retention)	none	none	

Despite the lack of organic abreaction we strongly advise against intramuscular or subcutaneous injection of the medium.

Our interest in this series lay in the frequency with which clear-cut, definite arm pain is experienced by patients receiving the rapid injection. There were, in all, 10 patients who described distress other than slight localized burning, in the series of 200 cases, or approximately 5 per cent (Cases 36, 38, 40, 50, 63, 64, 118, 129, 131, 190). The pain was felt, in most of them, in the shoulder rather than in the arm, or at least between the injection site and the shoulder. In our experience the pain was rarely severe, and special measures to combat it were not required. Contrasting these findings with the seemingly marked distress and the need for (or at least the use of) analgesics by mouth, heat applied locally, etc., when slow administration of the medium is used as reported by other workers, we have formed the conclusion that it is practical and wholly successful to administer neo-iopax as rapidly as possible. We go further and assert that by the use of this

technique it should be possible to limit all intravenous urography to this one apparently non-lethal medium and still dispense with the inconvenience of local pain which has been at times associated with it.

SUMMARY

The rapid intravenous administration of neo-iopax as a urographic medium in 200 cases is shown to eliminate all except an occasional, brief, initial, mild arm pain, and to make unnecessary the use of analgesic measures for such pain. The method recommended is to inject the solution through a 19 to 20 gauge needle in one minute or less, preferably within thirty seconds, with the arm extended and perpendicular to the trunk. No mortality or severe untoward reactions were encountered in this series.

We do not consider valid the usual list of "contraindications" to this medium as all of the numerous patients thus classified tolerated the administration of the medium without incident. No true contraindication existed in our series.

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PHYSICAL CHARACTERISTICS OF SOFT ROENTGEN RAYS

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INTRODUCTION

THE use of beryllium window roentgen tubes has expanded the roentgen-ray spectrum into regions not generally covered by existing literature. This paper is the report of an attempt to explore the physical characteristics of such radiation over the range of 30 to 100 kilovolts (peak). Any effort of this kind must of necessity be of a tentative nature because of the limited knowledge of certain elements involved. The least understood of these is probably the behavior of ionization chambers under such conditions. This is especially true of thimble chambers, which will probably go through some transition stages before a stage is arrived at wherein such instruments are as well understood and as reliable as is the case for their use with harder radiation. It may well be that chambers of the mesh type, the extrapolation chamber, or some future type of thimble chamber would have produced different results. Since none of these was available, the authors have attempted to obtain the best information with presently available facilities. Future developments in instrumentation may make it necessary to alter some of the information presented here. However, inasmuch as roentgen tubes of the type used for this experiment are coming into rather wide clinical and experimental use, it seemed worth while to present the best data that can be obtained under present conditions. That should at least make it possible for those working with such tubes to have some common information on which to base their work. Alterations can then be made as indicated by changes that may follow a fuller understanding of some of the elements involved.

EQUIPMENT

The equipment used throughout the period of about a year and a half during which these data were accumulated consisted of a self-rectified, self-contained, oil-immersed, 60 cycle unit, with a beryllium window tube having a window 2 millimeters thick and 1 inch in diameter. The usual precautions were taken for stabilizing voltage and tube current. The same precautions extended to timing apparatus and care in avoiding scattered radiation from supporting structures, room walls, etc.

INSTRUMENTS

It was felt that, so far as possible, the work should be done with some commercially available instrument, enabling its duplication by others interested in the problem. Accordingly, a 250 roentgen nylon chamber was obtained from the Victoreen Instrument Company. This chamber is commercially available and is designed to operate as part of the Victoreen condenser r-meter. The chamber was sent to the National Bureau of Standards for calibration against the Bureau's standard air chamber. The correction factors from this calibration were used throughout the investigation.

A standard air chamber was available for some of the work which had to be undertaken before a calibrated thimble chamber was available. It may be of interest to note that correction factors from 1.42 at 30 kv., unfiltered, to 1.10 at 100 kv. with 3 mm. of aluminum were necessary to bring this particular thimble chamber into agreement with the standard air chamber.

AIR ABSORPTION

In the use of radiation of conventional

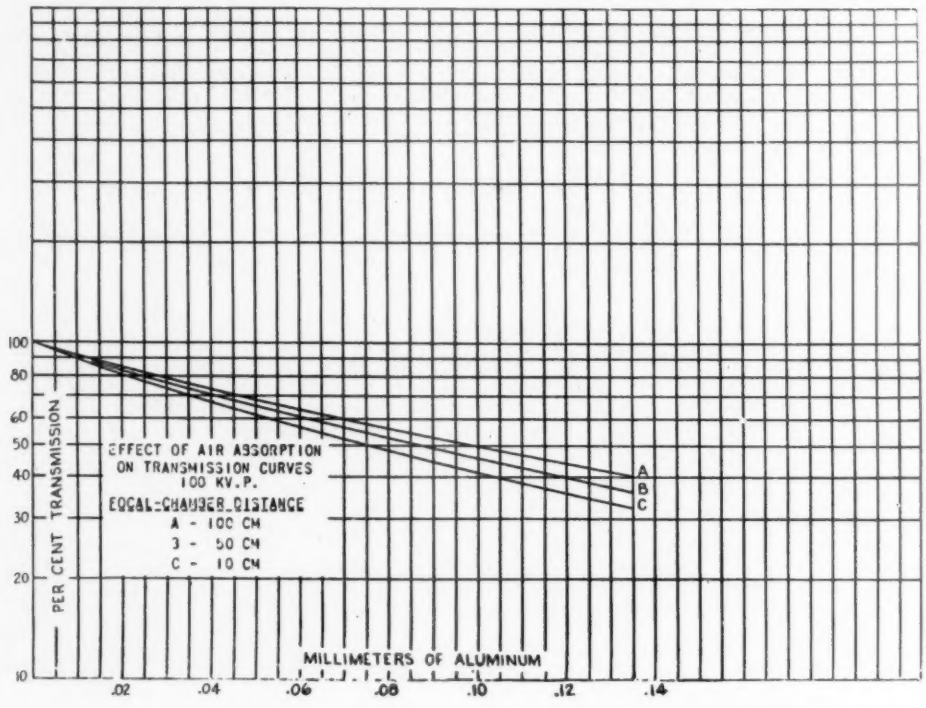

FIG. 1. Absorption curves in aluminum at 10, 50, and 100 centimeters.

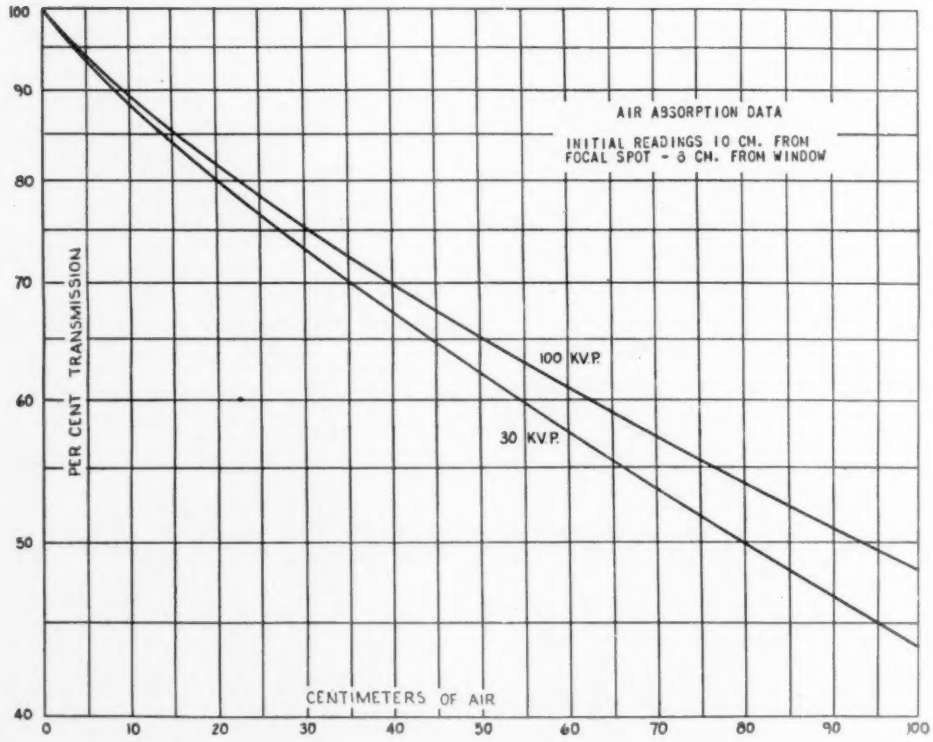

FIG. 2. Air absorption data.

quality, produced from glass-walled tubes at voltages above 60 kv. (peak), it has been general practice to ignore air absorption. That being the case, measurements taken at one distance can be computed for other distances by using the inverse-square law. This premise does not hold true when the inherent filtration is reduced to the point where the soft component of the beam is materially affected by absorption in a relatively short column of air. Fortunately, just as this paper was being prepared, a paper by Day and Taylor was published covering work on this problem for voltages from 7.5 to 200 kv. This excellent paper was concerned with focal-chamber distances of 119 to 319 centimeters. Inasmuch as the equipment which we were using permitted focal-chamber distances as short as 10 cm., we had explored the problem for distances of 10 to 100 cm. Although it is difficult to get a direct comparison between Day and Taylor's data and ours, due to the fact that they were using constant potential and a tube having a 1.5 mm. beryllium window, there would seem to be fair agreement between our data at 100 centimeters and that of Day and Taylor at 119 cm. when the differences in apparatus are taken into consideration.

Day and Taylor reported little change in the quality of the beam after it had been

TABLE I

AIR ABSORPTION CORRECTION FACTOR FOR FOCAL-CHAMBER DISTANCES OF 10–100 CM.

30 kv. (peak)—Minimum Filtration

From	To									
	10	12	15	20	25	30	40	50	70	100 cm.
10 cm.	1.00	0.98	0.94	0.88	0.84	0.80	0.73	0.67	0.58	0.46
12 cm.	1.02	1.00	0.96	0.90	0.86	0.82	0.75	0.68	0.59	0.47
15 cm.	1.06	1.04	1.00	0.94	0.89	0.85	0.78	0.71	0.62	0.49
20 cm.	1.13	1.11	1.07	1.00	0.96	0.91	0.83	0.76	0.66	0.52
25 cm.	1.19	1.16	1.12	1.05	1.00	0.95	0.87	0.80	0.69	0.55
30 cm.	1.25	1.22	1.17	1.10	1.05	1.00	0.91	0.84	0.73	0.57
40 cm.	1.37	1.34	1.29	1.20	1.15	1.09	1.00	0.92	0.80	0.63
50 cm.	1.49	1.46	1.40	1.31	1.25	1.19	1.09	1.00	0.87	0.69
70 cm.	1.72	1.69	1.62	1.52	1.45	1.38	1.26	1.15	1.00	0.79
100 cm.	2.17	2.13	2.04	1.91	1.82	1.74	1.59	1.45	1.26	1.00

100 kv. (peak)—Minimum Filtration

From	To									
	10	12	15	20	25	30	40	50	70	100 cm.
10 cm.	1.00	0.98	0.94	0.89	0.85	0.81	0.75	0.70	0.61	0.51
12 cm.	1.02	1.00	0.96	0.91	0.87	0.83	0.77	0.71	0.62	0.52
15 cm.	1.06	1.04	1.00	0.95	0.90	0.86	0.80	0.74	0.65	0.54
20 cm.	1.12	1.10	1.05	1.00	0.95	0.91	0.84	0.79	0.69	0.57
25 cm.	1.17	1.15	1.10	1.05	1.00	0.95	0.88	0.82	0.72	0.60
30 cm.	1.23	1.21	1.16	1.10	1.05	1.00	0.93	0.87	0.75	0.63
40 cm.	1.33	1.30	1.25	1.18	1.13	1.08	1.00	0.93	0.81	0.68
50 cm.	1.43	1.40	1.34	1.27	1.21	1.16	1.07	1.00	0.87	0.73
70 cm.	1.64	1.60	1.54	1.46	1.39	1.33	1.23	1.15	1.00	0.84
100 cm.	1.96	1.92	1.84	1.74	1.66	1.59	1.47	1.37	1.20	1.00

attenuated by 119 cm. of air. They pointed out that changes in the quality of the beam might be expected at distances less than 1 meter. Their prediction is in keeping with our findings. In Figure 1 are plotted three absorption curves in aluminum taken at 100 kv. (peak) at focal-chamber distances of 10, 50, and 100 cm. It will be noted that the half-value layers at these distances are 0.075, 0.086, and 0.10 mm. of aluminum, respectively. In Figure 2 are plotted absorption curves in air for focal-chamber distances of 10 to 100 cm. It should be noted that at the 10 cm. focal-chamber distance, there are only 6 cm. of air between the chamber and the window, since the focal-window distance was 4 centimeters.

If our data, taken between 90 and 100 cm. from the focal spot, are used to compute the linear absorption coefficient, the result is 6.76×10^{-3} cm.$^{-1}$ for 30 kv. and 5.73×10^{-3} cm.$^{-1}$ for 100 kv. These would compare with Day and Taylor's values of 6.1×10^{-3} cm.$^{-1}$ for 30 kv., and 5.1×10^{-3} cm.$^{-1}$ for 100 kv., where the inherent filtration was 1.5 mm. of beryllium plus 119 cm. of air, and where the tube was energized by constant potential.

In Table 1 will be found the correction factor for air absorption for 30 and 100 kv. (peak) for a tube having a 2 mm. beryllium window 4 cm. from the focal spot. If the inverse-square law is used to compute the intensity, the computed value must be multiplied by this correction factor.

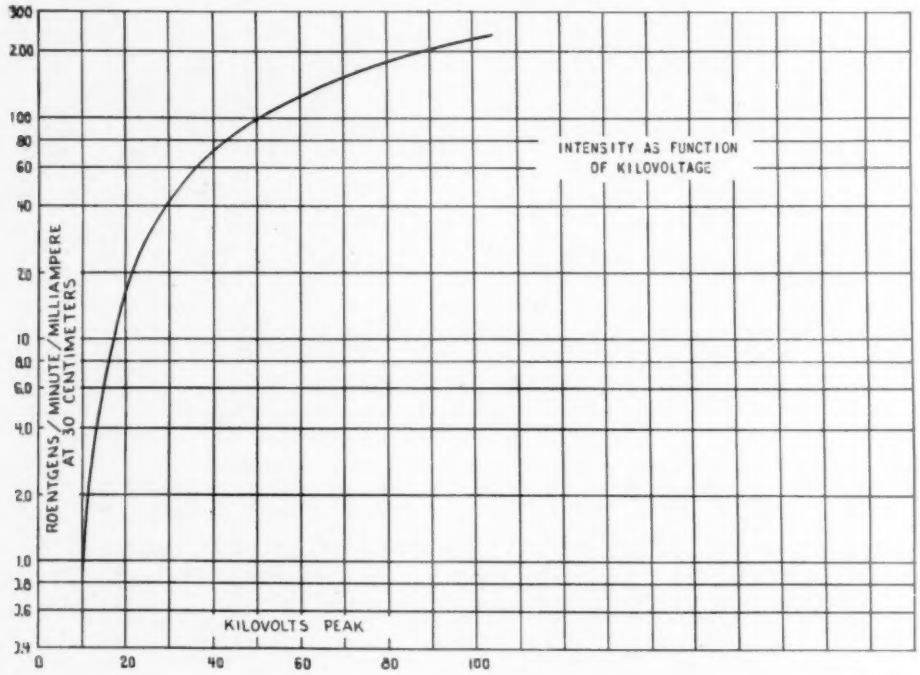

FIG. 3. Intensity as function of kilovoltage.

INTENSITY OF RADIATION

The intensity of radiation from tubes of this type is much higher than that encountered with tubes having glass windows. In Figure 3, the intensity at a 30 cm. focal-chamber distance is plotted for voltages from 10 to 100 kv. (peak). It will be noted that a very slight change in voltage between 10 and 30 kv. produces a great change in intensity. For that reason, at voltages below 30 kv. (peak) better than average conditions must prevail in the way of stable line voltage if the intensity is to

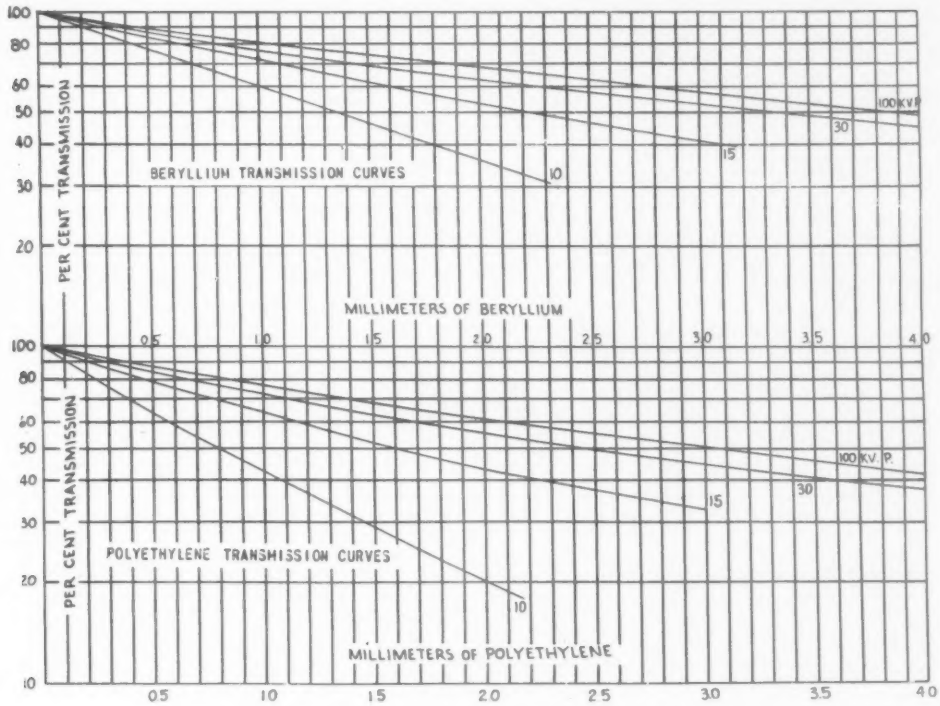

Fig. 4. Transmission curves in beryllium and polyethylene.

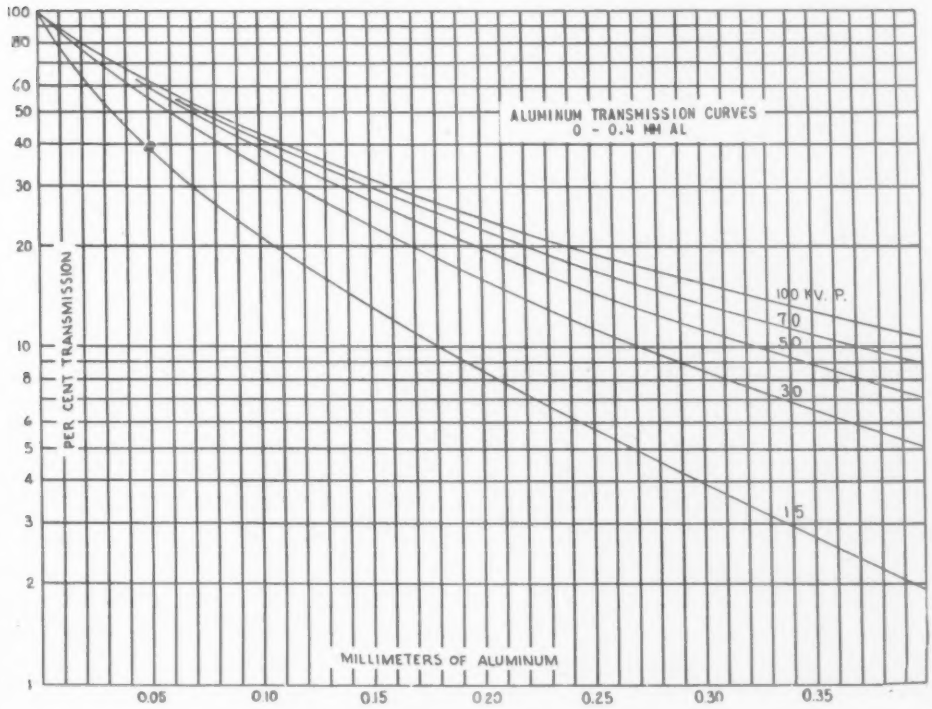

Fig. 5. Transmission curves in aluminum; 0–0.4 mm. aluminum.

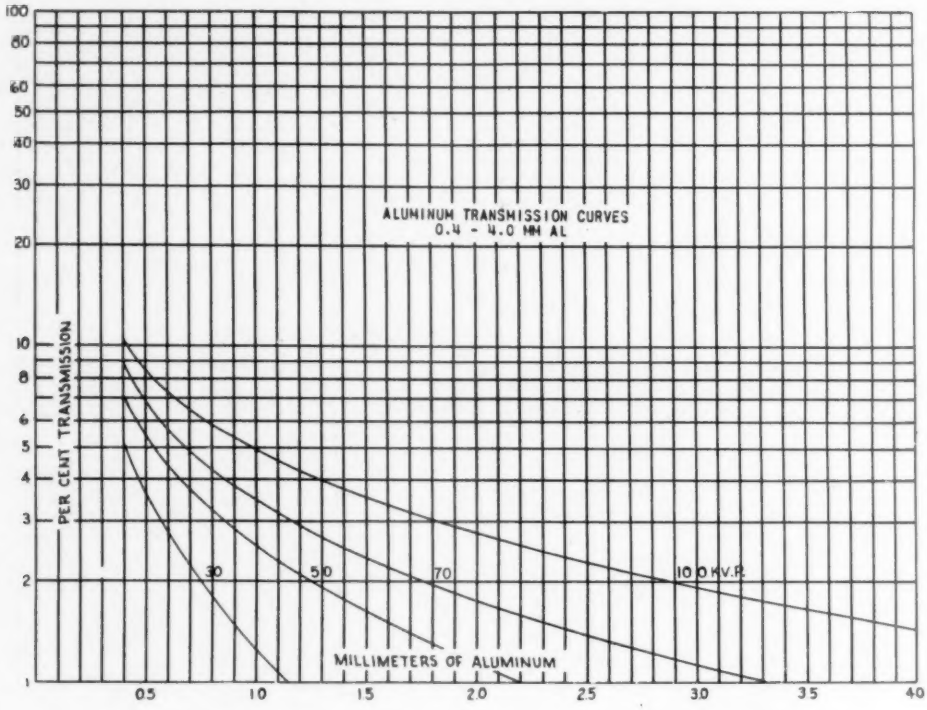

FIG. 6. Transmission curves in aluminum; 0.4–4 mm. aluminum.

TABLE II

QUALITY OF RADIATION

Kv. (peak)	Filter	Half-Value Layer (nominal)
10	Minimum	1.35 mm. beryllium
15	Minimum	2.21 mm. beryllium
30	Minimum	3.33 mm. beryllium
100	Minimum	3.85 mm. beryllium
10	Minimum	0.80 mm. polyethylene
15	Minimum	1.60 mm. polyethylene
30	Minimum	2.45 mm. polyethylene
100	Minimum	3.05 mm. polyethylene
30	Minimum	0.060 mm. Al
50	Minimum	0.070 mm. Al
70	Minimum	0.076 mm. Al
85	Minimum	0.078 mm. Al
100	Minimum	0.080 mm. Al
85	0.25 mm. Al	0.23 mm. Al
100	0.25 mm. Al	0.25 mm. Al
85	0.50 mm. Al	0.68 mm. Al
100	0.50 mm. Al	0.78 mm. Al
100	1.00 mm. Al	1.30 mm. Al
100	2.00 mm. Al	2.10 mm. Al
100	3.00 mm. Al	3.10 mm. Al

be controlled within workable limits for experimental or clinical work. That being the case, the greater part of this report will deal with voltages of 30 kv. or more.

TRANSMISSION DATA

One of the first difficulties encountered in taking absorption curves for very soft radiation is the matter of suitable filter material. Aluminum filters become so thin that it is difficult to be sure of uniformity, and it also becomes difficult to measure the thickness by conventional methods. Great care must be exercised in the selection of material of pure composition. One of the widely used aluminum alloys contains 4 per cent of copper. Filters of this material will give readings approximately 30 per cent less than filters of pure aluminum.

For certain laboratory work, it was felt advisable to take absorption data with something other than aluminum. Accordingly, beryllium and polyethylene were used. Finally absorption curves were plotted

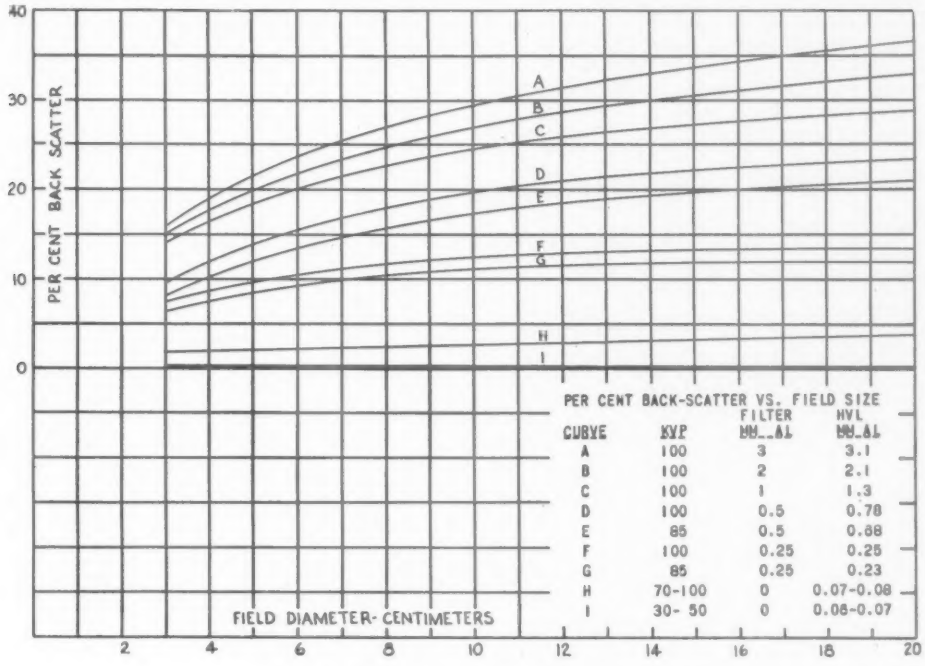

Fig. 7. Backscatter data.

in aluminum. This work was done using a standard air chamber at a distance of 30 cm. Transmission curves for beryllium, polyethylene and aluminum are shown in Figures 4, 5, and 6. From the transmission data, the nominal half-value layers were determined and are listed in Table II. The actual half-value layer may deviate slightly from these values at distances other than 30 centimeters. However, since distances

Table III

BACKSCATTER DATA

Kv. (peak)	Filter mm. Al	Half-Value Layer mm. Al	Per Cent Backscatter					
			3	5	10	15	20	cm. diam. field
			7.06	19.6	78.5	177	314	sq. cm. area
30	0	0.06	0	0	0	0	0	
50	0	0.07	0	0	0	0	0	
70	0	0.07	2	2	3	3	4	
85	0	0.08	2	2	3	3	4	
	0.25	0.23	6	8	11	11	12	
	0.50	0.68	8	12	17	20	21	
100	0	0	2	2	3	3	4	
	0.25	0.25	7	9	12	13	13	
	0.50	0.78	9	13	19	22	23	
	1.0	1.3	13	18	24	27	29	
	2.0	2.1	14	20	27	30	33	
	3.0	3.1	15	21	29	33	36	

of 10 to 30 cm. will be used for most clinical applications, the nominal half-value layers are probably adequate.

BACKSCATTER

In investigating backscatter and depth dose, it seemed advisable to use material approaching tissue as nearly as possible since work of other investigators had shown that some of the more common phantom materials, when used at low voltages, gave results that were not comparable with tissue. After considerable thought and experiment, it was decided to use beef. A section of lean beef 30 cm. in diameter and 7.5 cm. thick was used for backscatter measurements. A strip of tissue was removed, just permitting the chamber to be half buried. The refrigerating facilities that were available made it possible to maintain the moisture content of the meat at a uniform value. The results obtained were plotted in Figure 7 and arranged for ready reference in Table III. No attempt was made to consider backscatter of less than 2 per cent, since it was felt that the conditions under which the work was done did not make such figures significant.

The results were about as expected, with virtually no backscatter at the lower voltages. As voltages and filtration were increased, the backscatter became equal to that obtained with conventional sources. At higher voltage and filtration, the results are comparable with those of other investigators.

DEPTH DOSE

Sections of beef as described above were used for depth dose measurements. A 7.5 cm. thick section, 30 cm. in diameter was maintained below the thimble chamber at all times. A high-speed slicer was used to cut sections 1, 2 and 4 mm. in thickness. Combinations of these were used to obtain thicker sections. Care was taken to shape the tissue closely around the chamber at all times.

Repeated checks indicated that it was possible to leave the chamber in the electrometer without introducing appreciable error due to radiation penetrating the electrometer. This eliminated the errors brought about by repositioning the chamber in the tissue.

The data obtained are plotted in Figures 8 through 19. For comparative study, the

TABLE IV

DEPTH DOSE FOR 30 KV. (PEAK), NO FILTER (HALF-VALUE LAYER 0.06 MM. AL)

Depth mm.	Per Cent Depth Dose							
	1.77	7.06	19.6	44.1	78.5	177	314	sq. cm. area
	1.5	3	5	7.5	10	15	20	cm. diam.
	Focal-skin distance = 15 cm.							
0	100	100	100	100	—	—	—	
1	31	31	31	31	—	—	—	
2	20	20	20	20	—	—	—	
4	10	10	10	10	—	—	—	
8	4	4	4	4	—	—	—	
12	2	2	2	2	—	—	—	
	Focal-skin distance = 30 cm.							
0	—	100	100	—	100	100	100	
1	—	33	33	—	33	33	33	
2	—	21	21	—	21	21	21	
4	—	11	11	—	11	11	11	
8	—	4	4	—	4	4	4	
12	—	2	2	—	2	2	2	

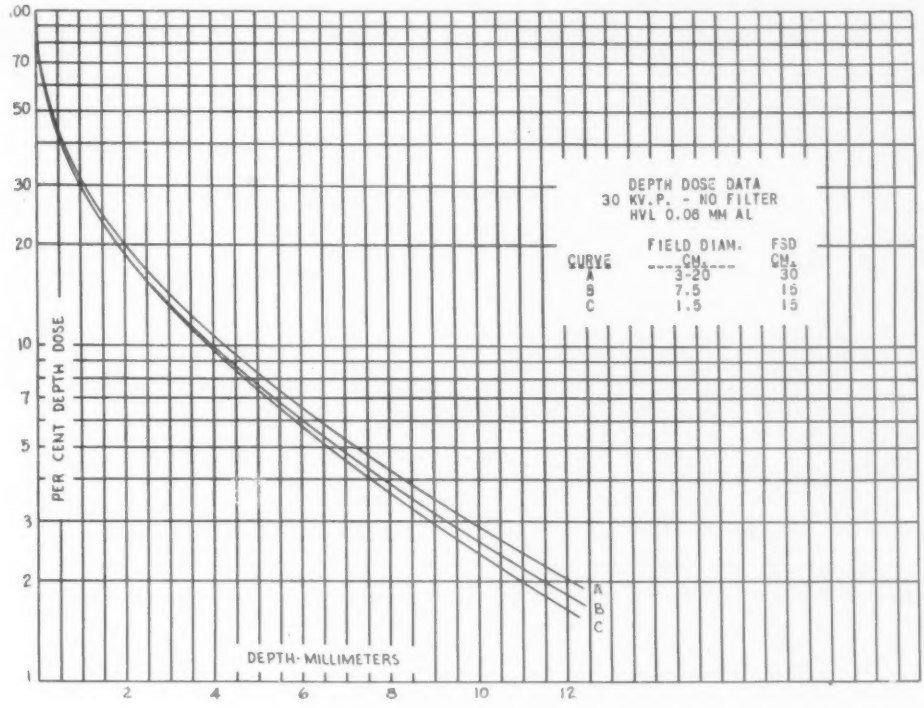

Fig. 8. Depth dose data; 30 kv. (peak); no filter.

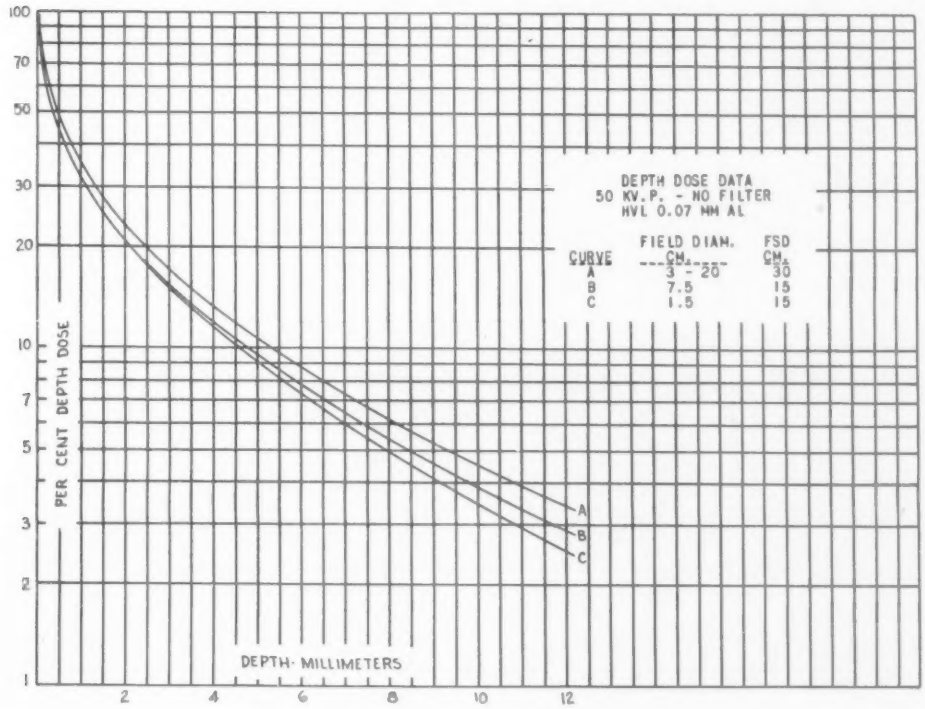

Fig. 9. Depth dose data; 50 kv. (peak); no filter.

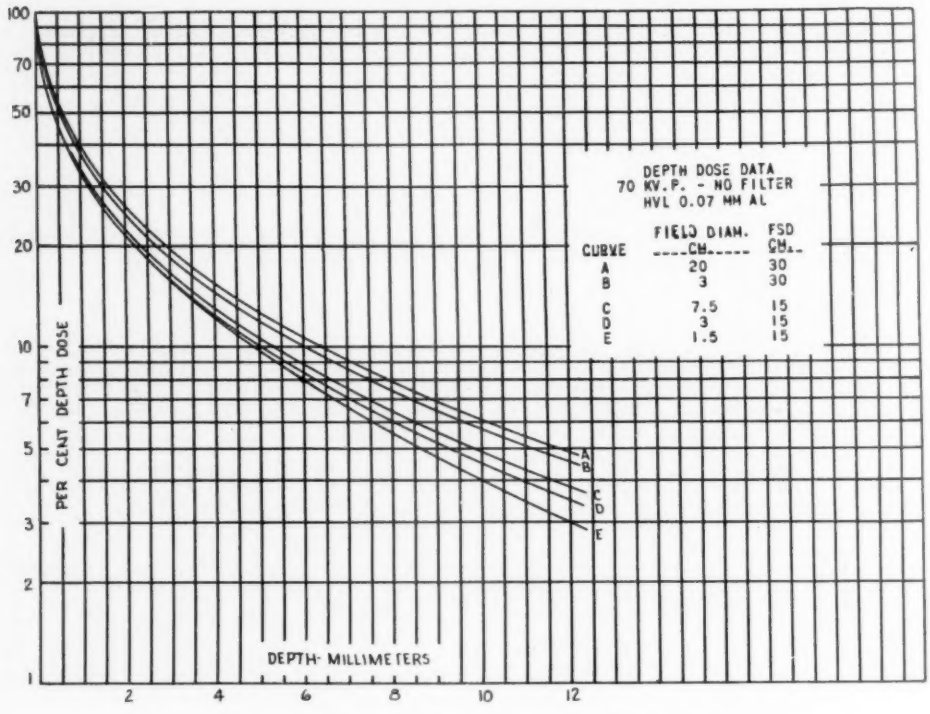

FIG. 10. Depth dose data; 70 kv. (peak); no filter.

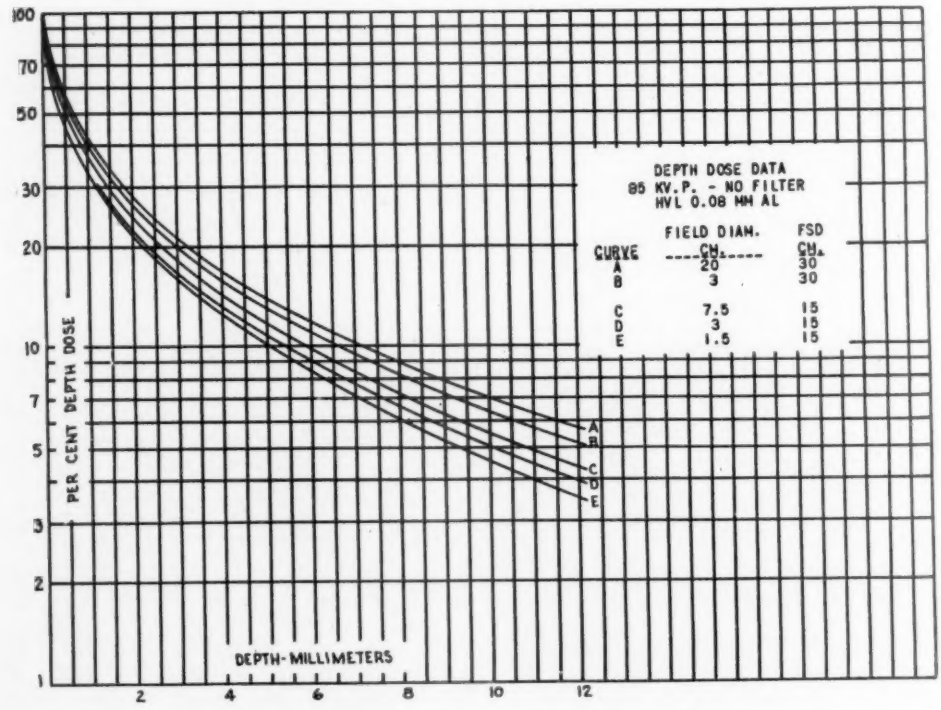

FIG. 11. Depth dose data; 85 kv. (peak); no filter.

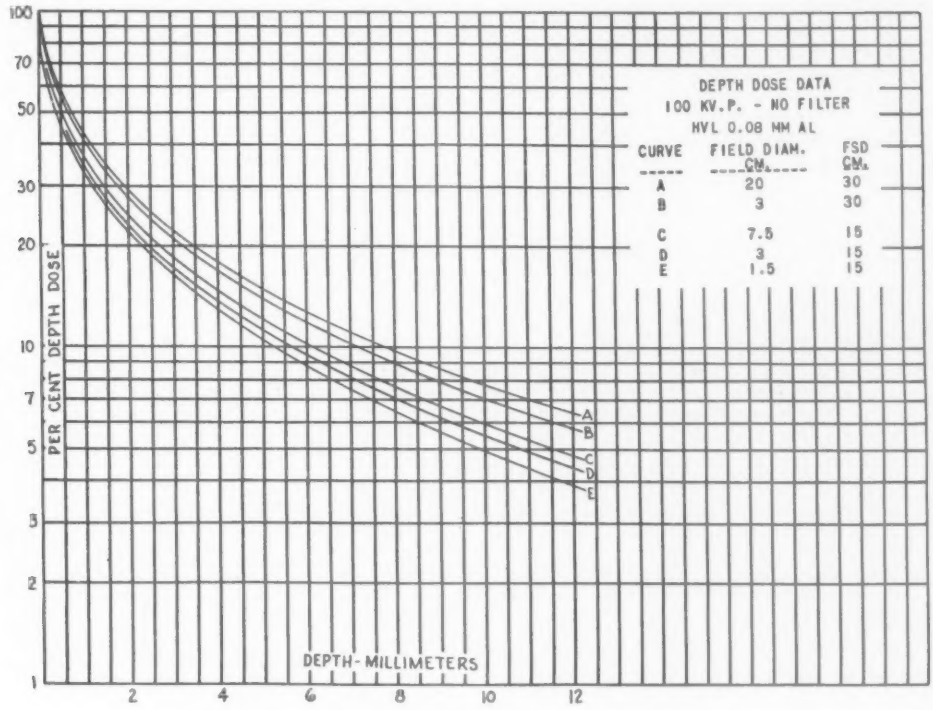

Fig. 12. Depth dose data; 100 kv. (peak); no filter.

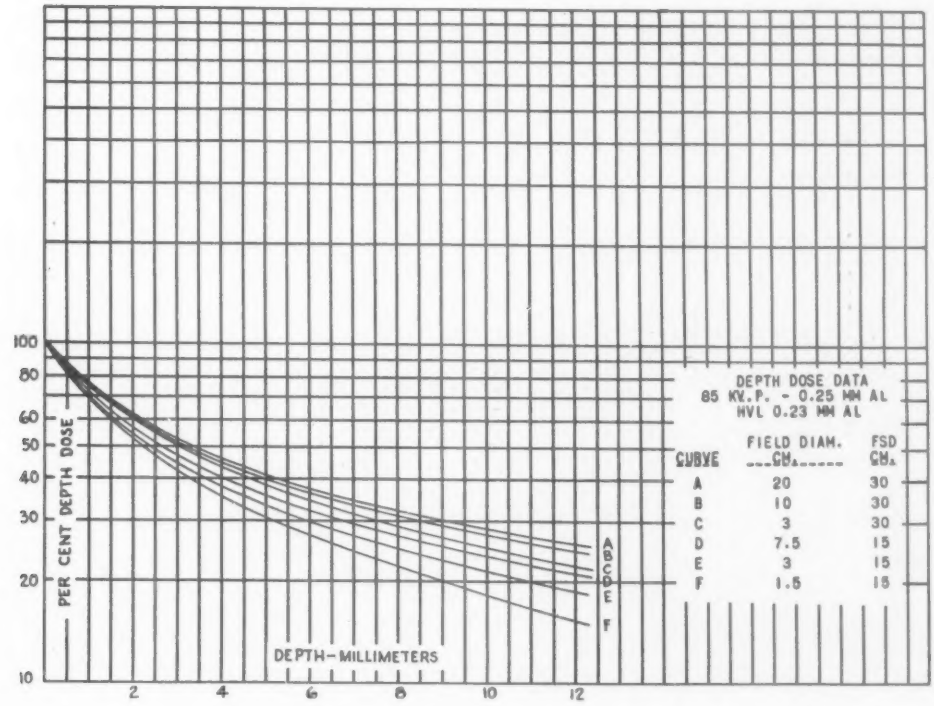

Fig. 13. Depth dose data; 85 kv. (peak); 0.25 mm. Al.

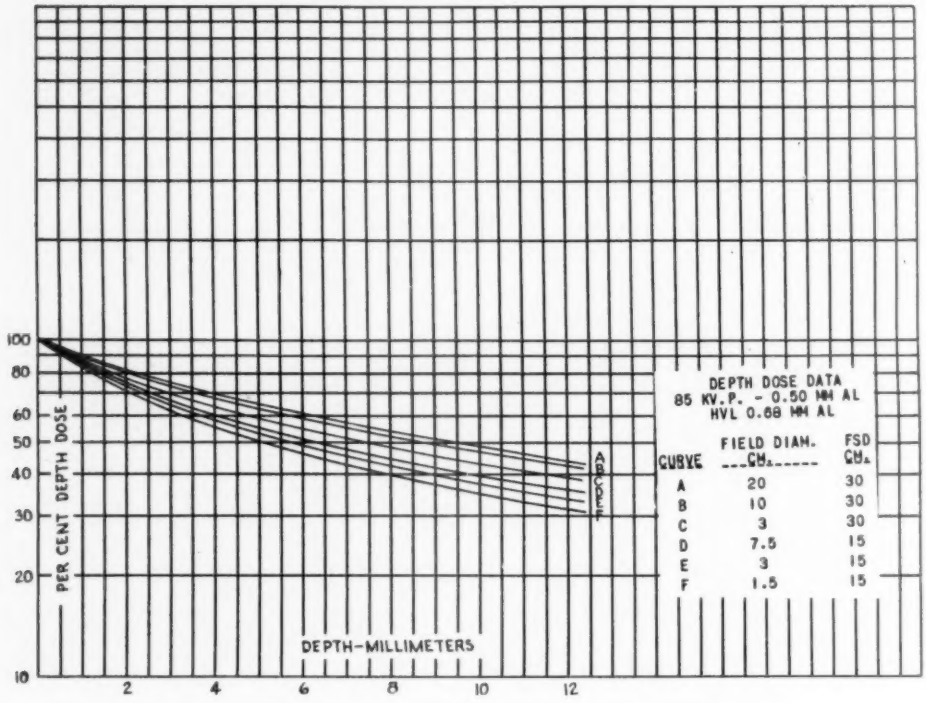

FIG. 14. Depth dose data; 85 kv. (peak); 0.50 mm. Al.

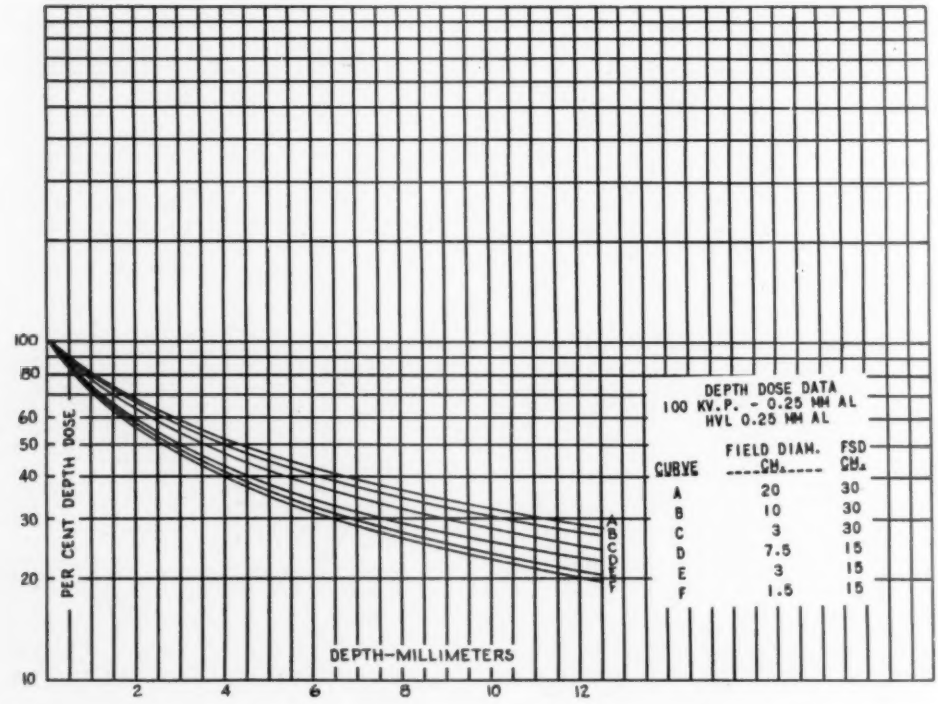

FIG. 15. Depth dose data; 100 kv. (peak); 0.25 mm. Al.

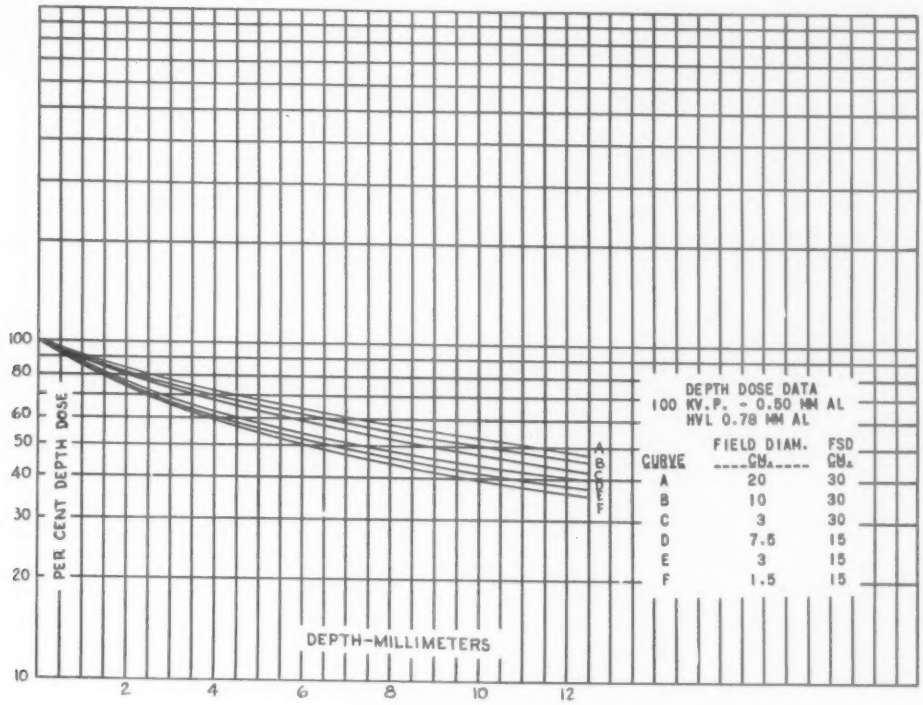

FIG. 16. Depth dose data; 100 kv. (peak); 0.50 mm. Al.

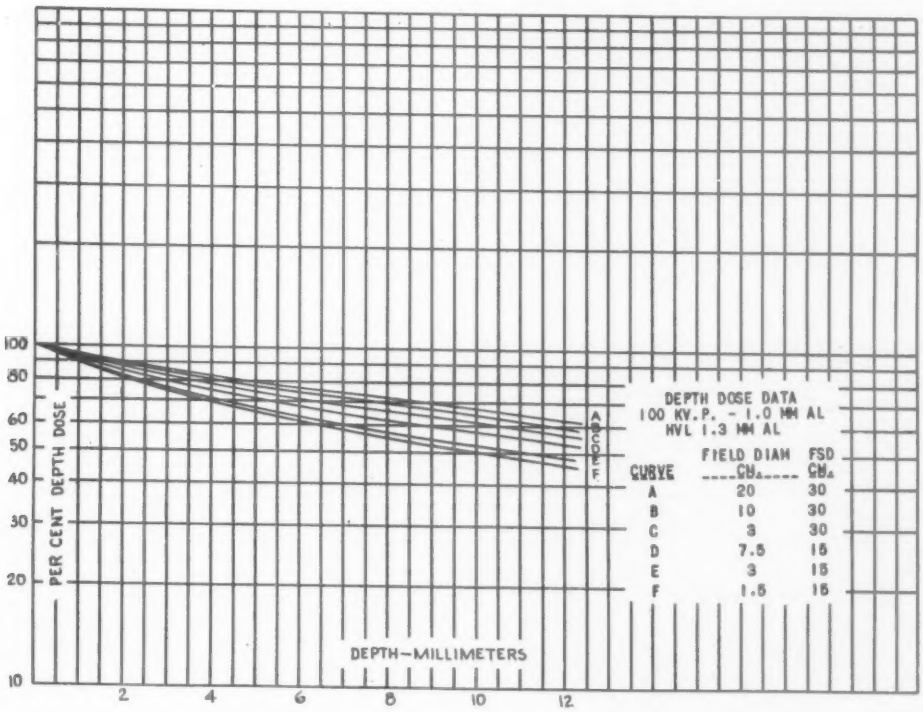

FIG. 17. Depth dose data; 100 kv. (peak); 1.00 mm. Al.

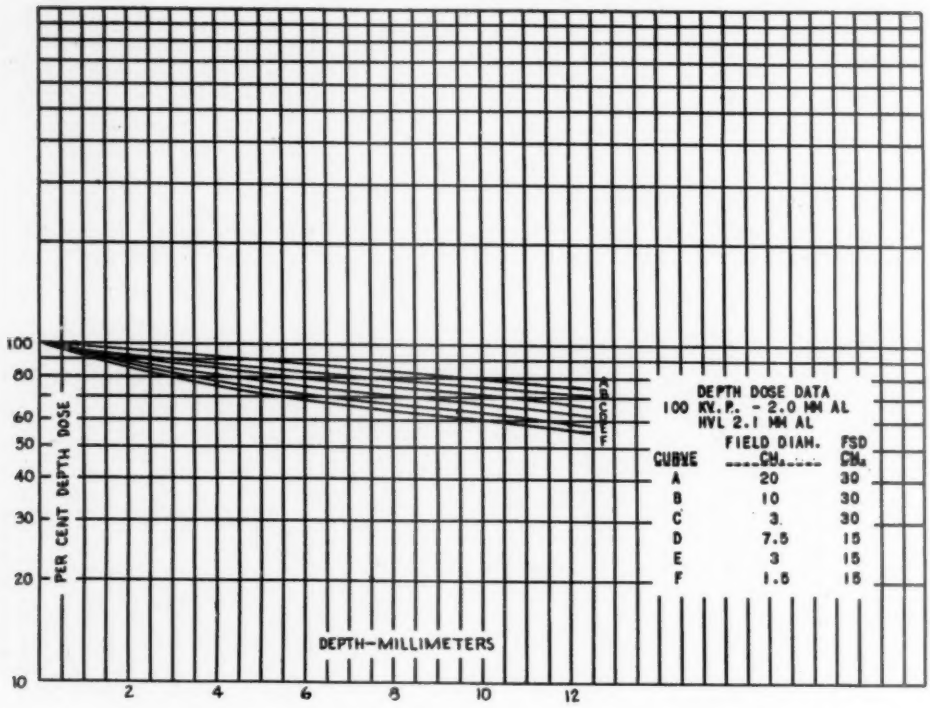

FIG. 18. Depth dose data; 100 kv. (peak); 2.00 mm. Al.

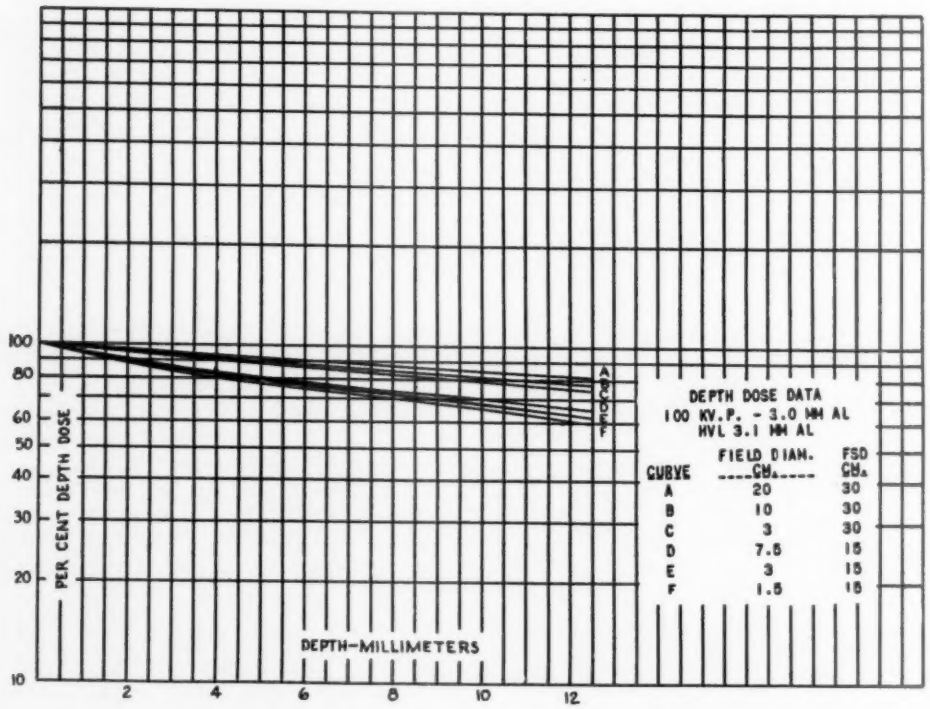

FIG. 19. Depth dose data; 100 kv. (peak); 3.00 mm. Al.

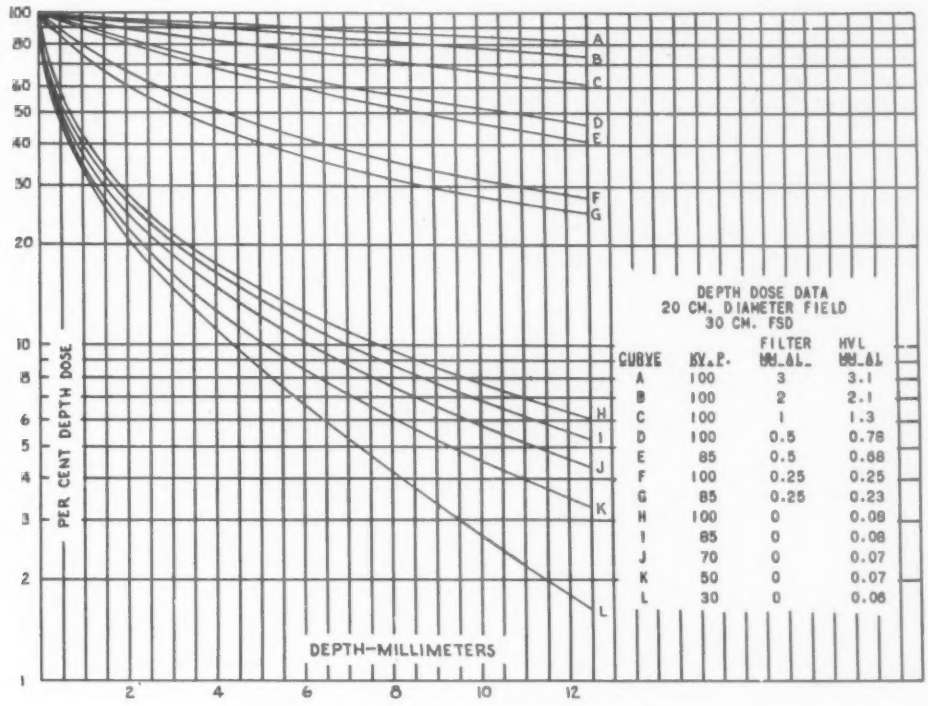
PER CENT DEPTH DOSE
DEPTH—MILLIMETERS
DEPTH DOSE DATA
20 CM. DIAMETER FIELD
30 CM. FSD
CURVE | KV.P. | FILTER MM. AL. | HVL MM. AL.
A 100 3 3.1
B 100 2 2.1
C 100 1 1.3
D 100 0.5 0.78
E 85 0.5 0.68
F 100 0.25 0.25
G 85 0.25 0.23
H 100 0 0.09
I 85 0 0.08
J 70 0 0.07
K 50 0 0.07
L 30 0 0.06

Fig. 20. Comparative depth dose data.

Table V

Depth dose for 50 kv. (peak), no filter (half-value layer 0.07 mm. Al)

Depth mm.	Per Cent Depth Dose							
	1.77	7.06	19.6	44.1	78.5	177	314	sq. cm. area
	1.5	3	5	7.5	10	15	20	cm. diam.
	Focal-skin distance = 15 cm.							
0	100	100	100	100	—	—	—	
1	33	33	33	33	—	—	—	
2	21	21	21	21	—	—	—	
4	12	12	12	12	—	—	—	
8	5	5	5	5	—	—	—	
12	3	3	3	3	—	—	—	
	Focal-skin distance = 30 cm.							
0	—	100	100	—	100	100	100	
1	—	35	36	—	36	36	36	
2	—	23	23	—	24	24	24	
4	—	13	13	—	13	13	13	
8	—	6	6	—	6	6	6	
12	—	3	3	—	3	3	3	

data for a 20 cm. diameter field at 30 cm. focal-skin distance over the entire quality range have been plotted in Figure 20. It will be noted that there is a considerable unexplored range between 100 kv. with no filter and 85 kv. with 0.25 mm. of aluminum. If clinical experience indicates that it is desirable to use some quality between

TABLE VI

DEPTH DOSE FOR 70 KV. (PEAK), NO FILTER (HALF-VALUE LAYER 0.07 MM. AL)

Depth mm.	Per Cent Depth Dose							
	1.77	7.06	19.6	44.1	78.5	177	314	sq. cm. area
	1.5	3	5	7.5	10	15	20	cm. diam.
	Focal-skin distance = 15 cm.							
0	100	100	100	100	—	—	—	
1	33	33	33	34	—	—	—	
2	21	22	22	3			—	
4	12	12	13	13	—	—	—	
8	5	6	6	6	—	—	—	
12	3	4	4	4	—	—	—	
	Focal-skin distance = 30 cm.							
0	—	100	100	—	100	100	100	
1	—	38	38	—	39	39	39	
2	—	25	25	—	26	26	26	
4	—	15	15	—	15	15	15	
8	—	8	8	—	8	8	8	
12	—	5	5	—	5	5	5	

those obtained with no filter and 0.25 mm. of aluminum, it would probably be advisable to resort to filters of some plastic material such as polyethylene to reduce the hazards associated with foil-type metal filters.

For ready reference, depth dose data for the conditions studied are arranged in Tables IV through XV. A rapid comparison of the depth dose for the various conditions can be had by reference to Table XVI, wherein is collected the depth dose 4 mm. below the surface for the various conditions.

TABLE VII

DEPTH DOSE FOR 85 KV. (PEAK), NO FILTER (HALF-VALUE LAYER 0.08 MM. AL)

Depth mm.	Per Cent Depth Dose							
	1.77	7.06	19.6	44.1	78.5	177	314	sq. cm. area
	1.5	3	5	7.5	10	15	20	cm. diam.
	Focal-skin distance = 15 cm.							
0	100	100	100	100	—	—	—	
1	34	35	35	36	—	—	—	
2	22	23	23	24	—	—	—	
4	13	13	14	14	—	—	—	
8	6	7	7	7	—	—	—	
12	4	4	4	4	—	—	—	
	Focal-skin distance = 30 cm.							
0	—	100	100	—	100	100	100	
1		40	41	—	42	42	42	
2	—	27	27	—	28	28	28	
4	—	16	16	—	17	17	17	
8	—	8	9	—	9	9	9	
12	—	5	6	—	6	6	6	

TABLE VIII

DEPTH DOSE FOR 100 KV. (PEAK), NO FILTER (HALF-VALUE LAYER 0.08 MM. AL)

Depth mm.	Per Cent Depth Dose							
	1.77	7.06	19.6	44.1	78.5	177	314	sq. cm. area
	1.5	3	5	7.5	10	15	2	cm. diam.
	Focal-skin distance = 15 cm.							
0	100	100	100	100	—	—	—	
1	34	36	36	37	—	—	—	
2	23	24	24	25	—	—	—	
4	13	14	14	15	—	—	—	
8	7	7	7	8	—	—	—	
12	4	4	5	5	—	—	—	
	Focal-skin distance = 30 cm.							
0	—	100	100	—	100	100	100	
1	—	42	43	—	43	44	44	
2	—	28	29	—	29	29	29	
4	—	17	18	—	18	18	18	
8	—	9	10	—	10	10	10	
12	—	6	6	—	6	6	6	

TABLE IX

DEPTH DOSE FOR 85 KV. (PEAK), 0.25 MM. AL (HALF-VALUE LAYER 0.23 MM. AL)

Depth mm.	Per Cent Depth Dose							
	1.77	7.06	19.6	44.1	78.5	177	314	sq. cm. area
	1.5	3	5	7.5	10	15	20	cm. diam.
	Focal-skin distance = 15 cm.							
0	100	100	100	100	—	—	—	
1	68	70	70	71	—	—	—	
2	52	54	55	56	—	—		
4	36	38	39	40	—	—	—	
8	23	25	26	27	—	—	—	
12	17	19	20	21	—	—	—	
	Focal-skin distance = 30 cm.							
0	—	100	100	—	100	100	100	
1	—	76	76	—	76	76	76	
2	—	61	61	—	61	61	61	
4	—	44	45	—	45	46	46	
8	—	29	30	—	31	32	32	
12	—	22	23	—	25	26	26	

TABLE X

DEPTH DOSE FOR 85 KV. (PEAK), 0.50 MM. AL (HALF-VALUE LAYER 0.68 MM. AL)

Depth mm.	Per Cent Depth Dose							
	1.77	7.06	19.6	44.1	78.5	177	314	sq. cm. area
	1.5	3	5	7.5	10	15	20	cm. diam.
	Focal-skin distance = 15 cm.							
0	100	100	100	100	—	—	—	
1	83	84	84	85	—	—	—	
2	72	73	74	74	—	—	—	
4	56	58	59	60	—	—	—	
8	40	43	44	45	—	—	—	
12	31	34	35	36	—	—	—	
	Focal-skin distance = 30 cm.							
0	—	100	100	—	100	100	100	
1	—	88	89	—	90	90	90	
2	—	78	79	—	81	82	82	
4	—	65	67	—	69	70	70	
8	—	49	51	—	53	53	54	
12	—	39	41	—	42	43	43	

TABLE XI

DEPTH DOSE FOR 100 KV. (PEAK), 0.25 MM. AL (HALF-VALUE LAYER 0.25 MM. AL)

Depth mm.	Per Cent Depth Dose							
	1.77	7.06	19.6	44.1	78.5	177	314	sq. cm. area
	1.5	3	5	7.5	10	15	20	cm. diam.
	Focal-skin distance = 15 cm.							
0	100	100	100	100	—	—	—	
1	71	72	72	74	—	—	—	
2	56	57	58	59	—	—	—	
4	40	41	42	43	—	—	—	
8	27	27	28	29	—	—	—	
12	20	21	22	23	—	—	—	
	Focal-skin distance = 30 cm.							
0	—	100	100	—	100	100	100	
1	—	78	78	—	79	80	80	
2	—	63	65	—	66	67	67	
4	—	47	49	—	50	51	51	
8	—	32	33	—	35	36	36	
12	—	25	26	—	28	29	29	

TABLE XII

DEPTH DOSE FOR 100 KV. (PEAK), 0.50 MM. AL (HALF-VALUE LAYER 0.78 MM. AL)

Depth mm.	Per Cent Depth Dose							
	1.77	7.06	19.6	44.1	78.5	177	314	sq. cm. area
	1.5	3	5	7.5	10	15	20	cm. diam.
	Focal-skin distance = 15 cm.							
0	100	100	100	100	—	—	—	
1	86	86	87	88	—	—	—	
2	75	76	77	78	—	—	—	
4	60	62	63	64	—	—	—	
8	45	47	48	49	—	—	—	
12	36	37	39	40	—	—	—	
	Focal-skin distance = 30 cm.							
0	—	100	100	—	100	100	100	
1	—	90	90	—	90	91	92	
2	—	82	82	—	83	84	84	
4	—	69	69	—	71	73	73	
8	—	54	54	—	56	58	58	
12	—	43	45	—	46	47	47	

TABLE XIII

DEPTH DOSE FOR 100 KV. (PEAK), 1.0 MM. AL (HALF-VALUE LAYER 1.3 MM. AL)

Depth mm.	Per Cent Depth Dose							
	1.77	7.06	19.6	44.1	78.5	177	314	sq. cm. area
	1.5	3	5	7.5	10	15	20	cm. diam.
	Focal-skin distance = 15 cm.							
0	100	100	100	100	—	—	—	
1	90	90	92	92	—	—	—	
2	82	83	86	86	—	—	—	
4	70	72	75	76	—	—	—	
8	56	58	61	62	—	—	—	
12	47	49	52	53	—	—	—	
	Focal-skin distance = 30 cm.							
0	—	100	100	—	100	100	100	
1	—	93	94	—	95	95	95	
2	—	88	89	—	90	90	91	
4	—	79	81	—	82	83	83	
8	—	66	68	—	70	71	72	
12	—	56	58	—	60	61	62	

TABLE XIV

DEPTH DOSE FOR 100 KV. (PEAK), 2.0 MM. AL (HALF-VALUE LAYER 2.1 MM. AL)

Depth mm.	Per Cent Depth Dose							
	1.77	7.06	19.6	44.1	78.5	177	314	sq. cm. area
	1.5	3	5	7.5	10	15	20	cm. diam.
	Focal-skin distance = 15 cm.							
0	100	100	100	100	—	—	—	
1	93	93	94	94	—	—	—	
2	87	88	89	90	—	—	—	
4	78	79	80	81	—	—	—	
8	65	67	69	71	—	—	—	
12	57	59	61	63	—	—	—	
	Focal-skin distance = 30 cm.							
0	—	100	100	—	100	100	100	
1	—	97	97	—	97	97	98	
2	—	94	94	—	95	95	96	
4	—	87	88	—	89	90	91	
8	—	76	78	—	80	81	82	
12	—	66	69	—	71	73	75	

TABLE XV

DEPTH DOSE FOR 100 KV. (PEAK), 3.0 MM. AL (HALF-VALUE LAYER 3.1 MM. AL)

Depth mm.	Per Cent Depth Dose							
	1.77	7.06	19.6	44.1	78.5	177	314	sq. cm. area
	1.5	3	5	7	10	15	20	cm. diam.
	Focal-skin distance = 15 cm.							
0	100	100	100	100	—	—	—	
1	94	95	95	95	—	—	—	
2	90	91	91	91	—	—	—	
4	82	83	84	84	—	—	—	
8	70	73	74	75	—	—	—	
12	60	63	66	67	—	—	—	
	Focal-skin distance = 30 cm.							
0	—	100	100	—	100	100	100	
1	—	97	98	—	98	98	98	
2	—	95	96	—	96	96	97	
4	—	90	92	—	92	93	94	
8	—	82	85	—	85	86	87	
12	—	75	78	—	79	80	81	

TABLE XVI

COMPARISON OF DEPTH DOSE DATA

Kv. (Peak)	Filter mm. Al	Half-Value Layer mm. Al	Per Cent of Surface Dose 4 mm. Below Surface: 1.5 cm. 15 cm.	20 cm. 30 cm. field diam. focal-skin distance
30	0	0.06	10	11
50	0	0.07	12	13
70	0	0.07	12	15
85	0	0.08	13	17
	0.25	0.23	36	46
	0.50	0.68	56	70
100	0	0.08	13	18
	0.25	0.25	40	51
	0.50	0.78	60	73
	1.0	1.3	70	83
	2.0	2.1	78	91
	3.0	3.1	82	94

SUMMARY

A study of the physical characteristics of the radiation from a roentgen tube having a 2 millimeter beryllium-to-air window and operating from 10 to 100 kilovolts (peak) is reported.

This report covers the instruments used, failure of the inverse-square law due to air absorption; change in quality due to air absorption at short distances; transmission curves in beryllium, polyethylene, and aluminum; backscatter data and depth dose information for radiation quality from a half-value layer of 0.06 millimeter of aluminum to 3.1 millimeters of aluminum.

It will be noted that with unfiltered radiation very little change in quality, backscatter, or depth dose is produced by a change in voltage. A marked difference in quality, backscatter, and depth dose is produced by the addition of a minimum amount of filter.

The depth dose information indicates the clinical possibilities of very soft radiation in those conditions where it is desirable to limit the radiation reaching underlying tissues.

The authors wish to express their appreciation to Mr. J. A. Victoreen, who made available a standard air chamber for some of the work, and to Mr. John P. Kelley, who assisted in the work on depth dose.

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DEPARTMENT OF TECHNIQUE

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ROENTGENOLGIC CONTROL OF URETERAL CALCULI ON THE OPERATING TABLE

By ERNEST NEWMAN, M.D., *and* LESTER NARINS, M.D.
From the Departments of Radiology and Genito-Urinary Surgery, Beth Israel Hospital
NEW YORK, NEW YORK

ONE of the hazards of surgery of ureteral calculi is the occasional inability of the surgeon to find the calculus or calculi after the ureter has been exposed surgically. It is known, for instance, that spinal anesthesia induces relaxation of the ureter, and occasionally after the administration of such an anesthetic, and in the movement incident to positioning, a calculus in one portion of the ureter, as determined by roentgen ray immediately prior to surgery, may shift either up or down in position, so that it cannot be found in the expected area. The portion of the ureter that can be exposed surgically is limited by the type of incision made for its approach. To make a lumbar incision, and then find that for the reasons mentioned above, the stone has slipped to the lower portion of the ureter and cannot be reached, is indeed an embarrassing situation. It was just such a case recently, in which one of two ureteral calculi was lost at the time of operation, that stimulated our interest in a method of roentgenologic control of ureteral calculi.

Prior to the fundamental work in roentgen control of calculi, done by Braasch and Carman,[2] the surgeon could depend only on his sense of touch to find a calculus or to determine if all the calculi present in the kidney had been removed. Quinby[3] and then Beer[1] used small films enclosed in sterile coverings, and took roentgenograms of the entire kidney after surgical exposure of this organ. However, after a search of the literature, we have been unable to find a report of a method by which the entire length of the ureter has been encompassed by a roentgen film on the operating table. The apparatus for such a film is standard and well known. The application of the method, however, is apparently new, and in suitable cases may be potentially of great value. By the method to be described, a simple abdominal film may be taken at any time during the operative procedure whether the patient is in the anteroposterior or lateral recumbent position.

FIG. 1. This shows the technique employed when the patient is in the lateral recumbent position. The assistant holds the 14 by 17 inch cassette, in front of which is the Lysholm grid. Sterility is maintained by covering the cassette, grid, assistants' hands and forearms with a sterile sheet, thus allowing the cassette and grid to be brought close to the patient. It is important in positioning the patient that the true lateral position be attained. If this is not possible, angling of the tube is essential.

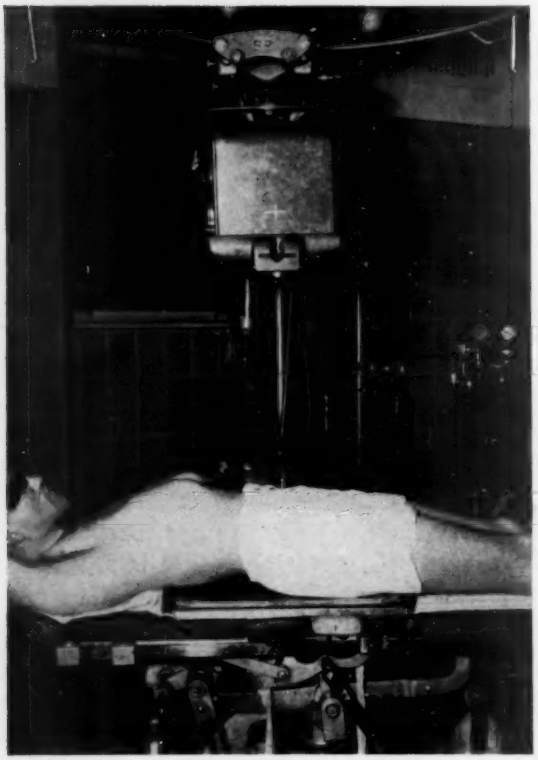

Fig. 2. The technique employed with the patient in the anteroposterior position. The wooden tunnel, loaded with the cassette and grid, is placed on the operating table prior to the operation.

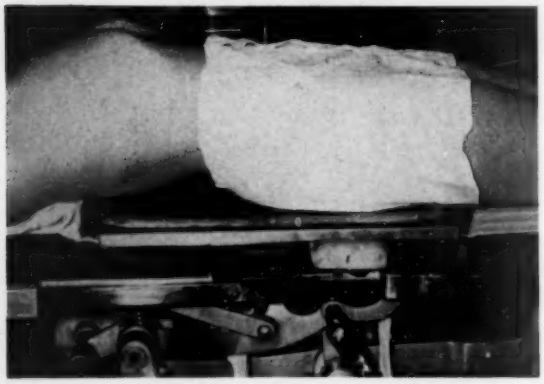

Fig. 3. A close-up of the loaded tunnel. The cassette may be exposed and exchanged during any part of the operative procedure without disturbing sterility. Films are developed and are available immediately for reading.

APPARATUS AND TECHNIQUE

The apparatus required for this method consists of a standard portable roentgen machine, a Lysholm stationary grid, and a wooden tunnel with a double slot for the grid and the cassette. The average factors used at this institution have been 25 ma., 85 kv., and two seconds. In all cases, the preoperative exposure is taken after the anesthetic has been administered, and the patient positioned on the operating table. It is important that explosive anesthetic gases should not be used when this method is employed. Figures 1, 2, 3, 4, 5 and 6 illustrate the technique and roentgenograms obtained.

Fig. 4. A preoperative roentgenogram made on the operating table after the patient had been positioned anteroposteriorly, and had been anesthetized. The roentgenogram shows an opaque shadow in the region of the right lower ureter.

Fig. 5. A roentgenogram made immediately after ureterolithotomy. A bougie has been inserted into the ureter from above. The surgical wound is still open, and sterility has been maintained.

Fig. 6. A preoperative exposure made on the operating table after the patient had been anesthetized and placed in the lateral recumbent position. The roentgenogram shows an opaque shadow in the region of the left upper ureter.

CONCLUSION

The purpose of this presentation has been mainly to re-emphasize the value of roentgen control in surgery of ureteral calculi. In such cases when a ureteral calculus cannot be found at the time of surgery the simplicity of the method described allows for the immediate use of roentgenologic control.

165 East 71st St.
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Thirty-second Annual Meeting: 1950, to be announced.

EDITORIAL

THE ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Fiftieth Annual Meeting of the American Roentgen Ray Society will be held at the Netherland Plaza Hotel, Cincinnati, Ohio, October 4–7, 1949.

This meeting of the Society promises to be a most important and scientifically profitable one. The selection of the Netherland Plaza Hotel as a meeting place is a splendid choice since the hotel facilities, as all the members of the Society well know, are ample to house the scientific and technical exhibits, and the rooms that are available for the instruction courses are unequaled. The Society returns to Cincinnati with pleasant memories of the previous meetings in that delightful city.

The program, which has been arranged for the Fiftieth Annual Meeting by President-Elect Portmann and his Committee, is a splendid one and should make a special appeal not only to the members of the Society but to all those attending the meeting. The program, which is published elsewhere in this issue, is evidence itself of the care that has been exercised in the arrangement of the scientific section. Doctor Portmann is to be congratulated upon having secured some of the leading medical men, internists, surgeons and radiologists to participate in this splendid program.

The Caldwell Lecture this year is to be held on Wednesday evening, October 5, and Doctor Portmann has selected Doctor Stanley P. Reimann, Director of the Research Institute, Lankenau Hospital, Philadelphia, Pennsylvania, to give the address. Doctor Reimann has chosen for his subject "The Differentiation of Cells and Tissues." This topic, needless to say, is of great importance to the medical profession and Doctor Portmann has made a happy choice in the selection of Doctor Reimann to give this important annual lecture.

The Instruction Courses this year are again under the directorship of Doctor Harry M. Weber. These courses, as always, are an important feature of the annual meetings, and the comprehensive courses which have been arranged by Doctor Weber are set forth elsewhere in this issue of the JOURNAL. Since the number who may attend these courses is of necessity limited, it is urged that those who anticipate taking such courses will fill out the order blanks at once in order to avoid any last minute confusion at the time of the meeting.

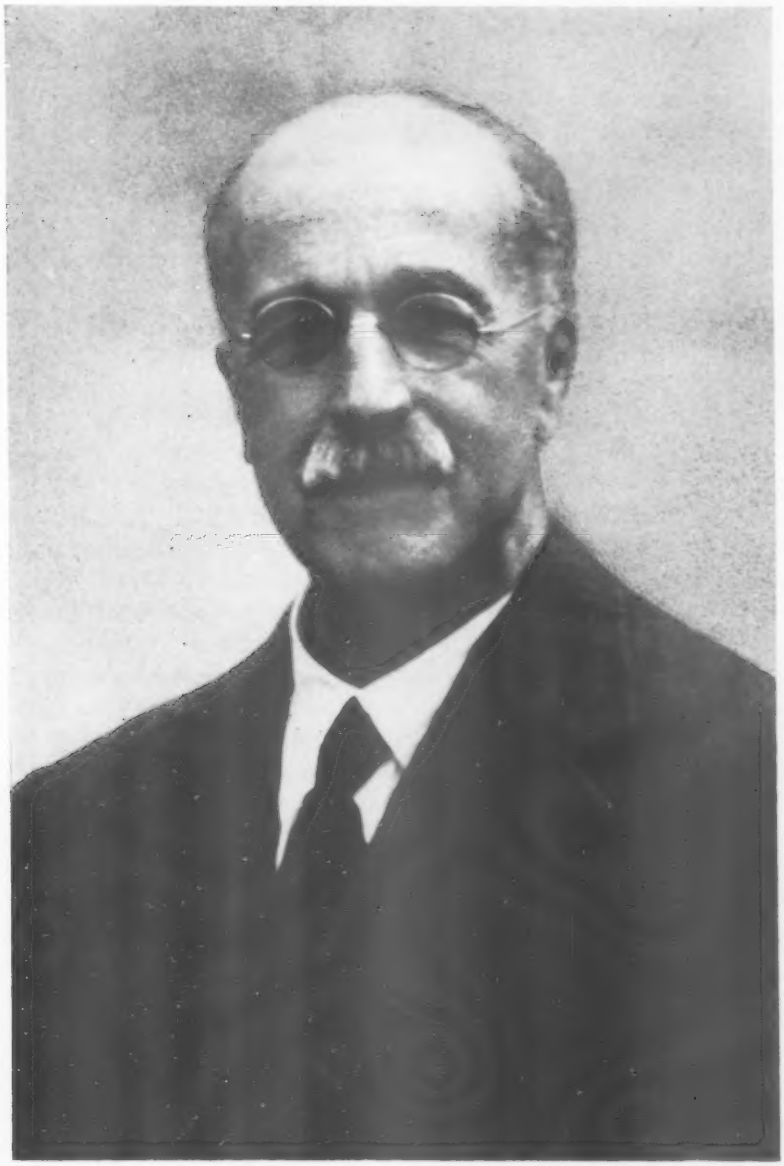
The Chairman of the Scientific Exhibit Committee is Doctor C. A. Good. He and his Committee have also arranged a superb exhibit covering many scientific problems of interest to radiologists, and some of the exhibits amplify and extend the papers that are to be given in the Scientific Program.

The Commercial Exhibit, which is one of the most important features of the annual meeting, is this year the largest that the Society has ever had, and the extent and scope of these exhibits should prove of greatest interest and profit to those attending the meeting. Fortunately, Doctor Portmann and his Program Committee have allotted a sufficient time during the morning and afternoon for viewing the Scientific and Commercial Exhibits.

The social functions of the meeting have not been neglected. The annual Golf Tournament for the Willis F. Manges Trophy will be held on Monday, October 3, at the

Kenwood Country Club. The annual banquet with well chosen entertainment will be held on Thursday evening; and the Ladies Entertainment Committee has arranged an excellent and varied program for the visiting ladies.

Thus, the forthcoming meeting in Cincinnati should stimulate much interest and will, undoubtedly, attract a large number of radiologists. In view of this, it is urged that all who anticipate attending the meeting make their reservations with the Netherland Plaza Hotel at the earliest possible moment.



ALFRED ERNEST BARCLAY

O.B.E., M.D. (Camb.), D.M. (Oxon.), D.Sc. (Hon. Oxon., and Manchester)
F.R.C.P., D.M.R.E., F.F.R.
1877–1949

DR. A. E. BARCLAY, "Barclay of Manchester," as he was known to a host of friends throughout the world, died at his home at Oxford, England, on April 26, 1949, at the age of seventy-two. His life was a long and useful one in the practice of medicine and radiology. He could look back over his shoulder at fifty years in which he

spent his life in and for radiology, though he was the sort of individual who rarely looked over his shoulder to the years, that had passed, but his thoughts were ever of the present and of the future of medicine and radiology and what part he might play in that field of endeavor to further extend knowledge into the realm of the unknown, and he bent his efforts toward solving some of the difficult problems in medicine and biology.

Alfred Ernest Barclay was born in Manchester, England, the son of Robert Barclay, on September 30, 1876, and was educated at Leys School and at Christ's College, Cambridge, and for his clinical studies he went to London, to the London Hospital, where after qualifying in 1904, he held various house appointments, and in his student days he became interested in radiology. While still at the London Hospital he became Accident Room Officer, and it was in this capacity while screening for innumerable needles and other foreign bodies that he began to appreciate the possibilities of roentgenologic investigation.

His first appointment as radiologist was as Clinical Assistant to Dr. Morton of the Electro-Medical Department and Clinical Assistant also to Dr. Sequeira of the Skin Department where treatment of ring worm was the main activity.

In 1906, having served such apprenticeship as was possible in those days of radiology at the London Hospital he went back to his native city of Manchester and in partnership with Dr. Bythell he set up in the private practice of radiology "to face the competition of a firm of chemists who had all of the x-ray work of the city." Following closely upon the heels of the opening of his office at Manchester he became Honorary Radiologist to the Ancoats Hospital and in 1909 he was appointed to organize an X-ray and Electrical Department in the new Manchester Royal Infirmary. There in the basement of that Infirmary with the crudest of apparatus he began his studies on the gastrointestinal tract. At the beginning of these studies he realized that it was the living anatomy that was of importance to the radiologist and that the "'dead' anatomy as was taught in the medical schools was wrong from every point of view." In his appreciation of the living anatomy as revealed in the roentgen studies, he began a campaign which he waged throughout his long life for a better knowledge and understanding of the normal structures as revealed by roentgenology, both from the anatomical and physiological viewpoints.

From his studies of the gastrointestinal tract he published a small monograph in association with Bythell on "X-ray Diagnosis and Treatment." This was published by the Oxford University Press in 1912. This small monograph attracted world wide attention and at once Barclay found himself among the forefront of those radiologists who were concentrating their energy and their ability in the roentgenological study of gastrointestinal problems. He was a great advocate of screen examinations of the gastrointestinal tract.

During World War I from 1914 to 1918 Barclay was commissioned as a Captain in the Royal Army Medical Corps and, with one colleague to assist him, was responsible for the x-ray service of a hospital unit which eventually grew to 35,000 beds. The supply of x-ray plates (glass plates were used in those days as negatives) was limited, consequently roentgenological work in the hospital unit consisted principally of screen localizations of foreign bodies and fractures.

Barclay tells how in those hectic days even with the press of work he found time to make a "bullet extractor, dental forceps with extractor prongs at right angles and a screen, unprotected, attached. The prongs were insulated and on meeting the foreign body they formed contact and rang a bell." In Barclay's hands it functioned well in recent cases but in long standing cases with the surrounding fibrous tissue the dissecting prongs were not strong enough for twisting and tearing fibrous tissue. In a letter to a friend he tells how he went across to France with this apparatus and demonstrated it,

but the cases were old long standing cases and the demonstrations he gave were far from convincing. This was a good thing, Barclay says, for the dangers from that type of tool were far from imaginary and hence it was not generally employed. He did, however, use the instrument at his own hospital and he recalled the use of it in the dramatic extraction of a small fragment of shell case from the lateral sinus of the brain.

Barclay was a prodigious worker in those days and he recalls how he ran the whole of the x-ray service of the Manchester Royal Infirmary single handed with the morning help of a technician and in the afternoon he carried on his private practice. Following which, he says, he was usually at committee meetings for the Red Cross for he was Assistant County Director, and in his small section of Manchester he organized twelve hospitals which gave bed accommodation and service for over a thousand patients. His inspections of these hospitals were mostly, as he said, "in the night watches when, with a swagger cane, he poked in the waste bins to check wastefulness."

Toward the end of the First World War he went to London for meetings and consultations to see what might be done to raise the status of radiology which in the Army was very low and with standards that gave tragic results clinically.

Seeing the need at first hand of better trained radiologists in those difficult days, in 1916, he thought that he might influence a Trust to endow a professorship and found an institute of radiology and he quite naturally turned to his old school, Cambridge, to survey the ground and receive encouragement for such an undertaking. But the hoped for finances did not mature. In all this endeavor he received the strong collaboration of such outstanding radiologists as Robert Knox, Sidney Russ, Cumberbatch, and others.

Barclay was not the sort of individual to be discouraged by one rebuff and in 1917 with some of his friends he persuaded the Cambridge authorities to start a course for a radiological diploma. He tells us that all these negotiations went well but very slowly. The University (Cambridge) was a little hesitant about starting such a course but with Barclay's personal check handed over to the University to guarantee it against loss the negotiations for such a school for a radiological diploma came to fruition in 1920 when the first course opened. Barclay's enthusiasm for the course, bolstered by his friends and lecturers who worked without reward, made the course a popular one and no University funds were needed to carry on the courses.

The Manchester Royal Infirmary in 1918, recognizing the tremendous value of Barclay's work and the increasing status of radiology in the medical world, appointed him to the Honorary Staff. This was a long step in the advancement of radiology and particularly the advancement of Barclay himself for not only could he enter into the clinical life of the hospital with the full responsibilities that that implied and which he so ardently sought but in addition he had the backing of his colleagues and of the lay board in working for a new and more adequate department. This was realized in 1921 and shortly afterwards Barclay was joined on the staff at the Manchester Royal Infirmary by Dr. G. M. Woodburn Morison.

It was during his years in Manchester that Barclay gained world wide recognition not only by his contribution to the medical literature dealing with radiological problems but by his ceaseless endeavor for the advancement of radiology in particular and medicine in general. He not only gained a world wide audience in the publication of his papers but his engaging personality and his capacity for friendships brought him a host of personal friends throughout the world.

In 1928 Barclay, at the height of his splendid position and professional standing in his native city of Manchester, decided to go to Cambridge to carry on the radiological teaching for the D.M.R.E. The altruistic spirit which Barclay exhibited in this decision was an example of the fine atti-

tudes which he displayed throughout his long professional life. Manchester was his home; here he had established his professional reputation but his great interest in the diploma course was something to which he had given much thought and with the death a year earlier of Shillington Scales the diploma course was in danger of lapsing. This decided Barclay. Cambridge University could offer little salary and, as Barclay says, not a penny for the running of the department, and for secretarial assistance and other expenses Barclay paid out of his own pocket, but the opportunities for research which such an association presented overcame these "minor" financial responsibilities. He began at once to build up techniques of examination, particularly direct cineroentgenography. It was an attempt to study the mechanism of swallowing that led to Barclay's great desire to develop this type of technique. It was Barclay's desire and intent to establish at Cambridge a well equipped radiological department functioning not only as a department of radiology but serving the biological laboratories as well. But the authorities at Cambridge saw no need for such a department and were not prepared to support the brilliant plans that Barclay envisaged. However for nine years he worked diligently building high the prestige of the Cambridge Diploma. In 1937 Cambridge University decided to abolish all diplomas and there was no place therefore for Barclay or his department in Cambridge.

At this time Janker of Bonn and Russell Reynolds were showing good results with indirect cineroentgenography. These results were shown in Oxford and half the newly acquired funds for the Nuffield Institute for Medical Research were allotted to explore this technique and Barclay was invited to join Dr. K. J. Franklin in this work which was his conception.

It was at Oxford University in the Nuffield Institute for Medical Research that Barclay's bent for mechanical gadgeteering was given full sway. He found that the apparatus which Janker and Russell Reynolds had elaborated was only capable of operating vertically and hence was useless for animal work. He soon constructed a horizontal couch and a mechanism for taking direct serial roentgenograms. With this improved but inadequate setup Barclay and his coworkers did their experiments on the mechanism of dust excretion from the lungs. It was while working on this problem that Sir Joseph Barcroft asked for cooperation in his problem of what happened to the ductus arteriosus at birth and Barclay tells us "that we had a couple of years of delightful cooperation with him. The work was done on sheep fetuses. It was number 300 odd on which we started for Barcroft had been on the problem for years." For this particular type of work Barclay and his collaborators developed special techniques and by extraordinary luck at last got the direct cineroentgenographic machine to work satisfactorily. "We solved Barcroft's problem out of hand as soon as we learned to interpret the exceedingly complex shadows given by the thorotrast as it canalized the heart and main vessels. We settled the problems of the ductus as to how and when it closed and in doing so we, quite incidentally, obtained incontrovertible proof of the working of the foetal circulation as a whole and the changes that occur when adult circulation is established—a 300 year old controversy!" This work was published in 1944 in book form.

The latter part of this work was carried on during the imminence of war and Barclay in his official capacity as Adviser in Radiology to the Ministry of Health was responsible for the organization of the whole civilian x-ray service of the country and in such a capacity there was little time for research. With the rapid expansion of the hospital facilities to care for the wounded and sick it became Barclay's responsibility to see that each of these hospitals had properly equipped departments with such equipment as was available in those hectic days, and not only must these departments be equipped but supervised by a much reduced number of radiologists.

This however was accomplished with Barclay's usual thoroughness, and considering the conditions, the standards of roentgenray services were surprisingly high. In his capacity as Adviser to the Ministry of Health, Barclay travelled the whole country from end to end visiting all these hospitals and seeing all the radiologists in their home working conditions. He found it extremely interesting and quite invaluable work but it must have made great inroads upon his own physical well being, though he never admitted it.

It was in 1940 that Trueta and Barnes came to discuss with him the problem of uremia following traumatic crushed limbs. But while there was much discussion concerning the problem and it offered an intriguing type of investigation as well as an interesting one, the press of war and the conditions which it posed prevented any serious approach to the problem until 1945. As soon as the war was over, they started work on this problem. They were in possession at this time of a well equipped department at the Nuffield Institute and it was easy to adapt the techniques that had been employed for the fetal circulation work, and again roentgenology was the spearhead of attacking the problem and direct cineroentgenography was the key technique which gave exact timing of the flow. Barclay says, "The story of this, the most exciting and satisfying research in which I have ever taken part, is told in the book of renal studies in which we brought forward the conception of the peripheral vascular supply as virtually an organic structure spread through the kidney, and likely spread throughout the whole body on much the same plan, to regulate the supply of blood to organs on a functional basis. In the later stages of this work the work passed out of the range of radiology into histology. Could radiology help in this as in other branches?" Barclay tells us he set to work on this problem and within a few weeks demonstrated to his own satisfaction that given suitable equipment "I would be able to show even capillaries on radiographs. Moreover I would not only be showing them as under a microscope but in a big field in which capillaries and other vessels would be seen over considerable lengths and we should get some sense of the relationship of small vessels to large vessels." And out of this work came his important paper "Micro-arteriography."*

Barclay, as ever intrigued with interesting problems of research and their possibilities, thought of the important "side line" that has evolved out of this technique in the incursion into the field of botany and he published his interesting observation from this study in the *British Journal of Radiology*.

Following a major operation two years ago Barclay realized that he was perhaps living on borrowed time and with characteristic courage in spite of his lessening energies he drove himself with added interest into his work on microradiography and in conjunction with F. H. Bentley there resulted the discovery of a circulatory shunt mechanism in the wall of the stomach,† thus proving his belief that the "peripheral vascular supply which he observed in his studies on the kidney was most likely spread throughout the whole body on much the same plan to regulate the supply of blood to organs on a functional basis." Thus, among the last of Barclay's observations was a magnificent piece of research carried on by a man whose days and length of life were numbered.

In the winter of 1948 he went to Torquay but realizing as he did the short space of time remaining for him, he took his laboratory with him and erected it in a "back bedroom only 20 yards from his own bedroom." He says it was a "delightful little laboratory and well equipped and more convenient than what I have in Oxford." But his strength was far spent and he says he could do nothing even under those easy conditions. "Luckily," says he, "I had an A-1

* *Brit. J. Radiol.*, 1947, *20*, 394–404; Am. J. Roentgenol. & Rad. Therapy, 1948, *60*, 1–12.

† See Editorial, "Gastric vascular shunts." Am. J. Roentgenol. & Rad. Therapy, April, 1949, *61*, 561–564.

laboratory assistant who did things for me and I did get some bit of result that made the whole move well worth while." He found that even with such an outfit as he had established in his back bedroom he was able to show the nuclei of cells.

His last paper dealing with the occurrence of hematite deposits was published in the May, 1949, issue of the *British Journal of Radiology*. In a letter written on April 1, 1949, he says apropos of this work, in spite of his enfeebled condition, of which he complains bitterly: "Yet only 2 days ago I did a trick and sent a page to the B.J.R.—a lucky last fluke of an exposure I did at Torquay. It shows iron absorbed into and around the nuclei of the lung cells in a haematite miner—it was the only specimen I happened to have at Torquay—if I'd used normal tissue I'd have drawn a blank, as my assistants did when they tried to repeat when the apparatus was reinstalled here [at Oxford]. So luck holds right up to the end. And what a lot of fun I've had out of 50 years in radiology!"

In these last days Barclay saw his greatest triumph for in these few months he made epoch-making contributions to physiology as well as to pathology by the use of roentgenological methods, and one recalls a statement which he made in a recent letter: " . . . discoveries lie all round for any man who has the sense and adaptability to take radiology into physiological, pharmacological, zoological and botanical research." One of America's greatest physiologists in commenting on Barclay's studies in which he employed microradiography says that he thinks "the technique is one of the greatest milestones in medical science."

Barclay was in turn President of the Röntgen Society, of the British Institute of Radiology, and of the Electrotherapeutics Section of the Royal Society of Medicine. In 1931 he was appointed Silvanus Thompson lecturer. He had been chosen to deliver the Mackenzie Davidson Memorial Lecture under the auspices of the British Institute of Radiology in March, 1949, but when the time came he was far too ill to do so, and the Institute as a mark of respect to him met only for formal business, arranging no other event to take the place of the expected lecture. Its president travelled to Oxford to present him with the lecturer's medal. Barclay was an honorary member of the American Roentgen Ray Society and of the Australian and New Zealand Association of Radiology, and an honorary fellow of the American College of Radiology; he was a corresponding member of the Radiological Society of North America.

Barclay was the author of innumerable articles published in scientific journals throughout the world and the author and co-author of several books. In addition to his first radiological publication "X-ray Diagnosis and Treatment" published in 1912 in collaboration with Bythell, his textbook on the "Digestive Tract" appeared in 1933 and there was a second edition published in 1936. His studies with Trueta on the renal circulation were fully published in book form in 1947.

In 1906 Dr. Barclay married Miss Mary McFarlane and what a happy choice he made! Barclay himself attributed what success he might have had to the help and ability of Mrs. Barclay who looked after everything in his life outside of his medical specialty, thus leaving him free to direct his creative faculties, and those who have been privileged to visit the Barclays' home will recall pleasant memories of a charming host and hostess.

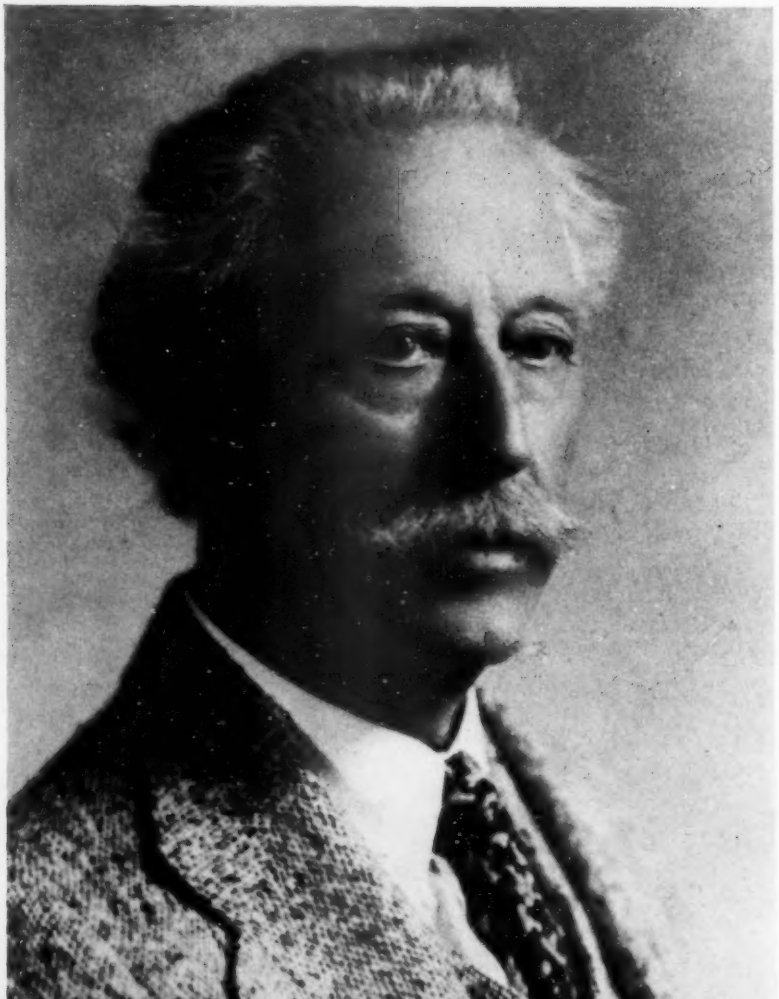
Barclay was the recipient of many honors and gave many memorial and other lectures both in England and abroad. Among his latest honors and perhaps the ones he valued most were the awards of the Honorary D.Sc. by both Oxford and Manchester Universities. In March, 1949, Barclay received the Bose Memorial Gold Medal as the first Lecturer (in absentia) in memory of the outstanding Indian scientist Sir Jagadish Bose. He was at the time of his death President Emeritus of the Sixth International Congress of Radiology which is to be held in London in 1950 and he had a unique record of an unbroken attendance at all of the

past five International Congresses of Radiology. Barclay will be sorely missed at the forthcoming meeting in his own country.

He has a host of friends and acquaintances throughout the world who mourn his death. During his long life, in spite of the great honors that came to him and the magnificent work which he had done, he never lost the common touch and he made friends and inspired individuals wherever he went and it was a fitting tribute to a worthwhile life that prompted Oxford and Manchester Universities to confer upon him one of their most coveted gifts. Their stamp of approval in conferring such an honor was a just tribute to one whom the medical world has known over the years as being one of the "greats" in medicine. His friends and admirers were just as numerous in this country as in his own native England and one might paraphrase the oft repeated statement that "there will always be an England" by saying that "there will always be a medical England as long as it produces men of the stamp of Barclay." He stands with Hunter, Bright, Hodgkin and a whole host of others in England who have always attracted the medical minds of the world.

Barclay's indomitable spirit and search for knowledge carried him through years which would have daunted men of lesser soul and by such an example he sets a challenge to us elders and an inspiration to the youth of the world.

L.R.



HENRY HULST
1859–1949

THE death of Dr. Henry Hulst in Grand Rapids, Michigan, on January 2, 1949, writes finis to one of the most noteworthy careers in radiology. The roentgen diagnosis of tuberculosis probably owes more of its foundation to Henry Hulst than to any other American radiologist, in that he was the first to roentgenograph the chest in one second or less; and this without the relatively unsatisfactory intensifying screens of 1903. Older radiologists will remember that M. K. Kassabian of Philadelphia was an able pioneer, a martyr to the deadly quality of the unknown for which the "x" still stood in "x-ray." In December, 1903, at the Philadelphia meeting of the American Roent-

gen Ray Society, Dr. Kassabian showed, and later published, a roentgenogram of a chest showing large tuberculous cavities, made in three installments of ten seconds each, while the patient held his breath, and breathed between times. This was a great improvement on the then standard plates (for roentgenograms were on glass plates until the demands of the First World War were met with films) made while the patient breathed. At this same meeting, Dr. Hulst showed plates made in a second or less, of a diagnostic quality that would pass today. (He showed them in Chicago in 1902!) The Transactions of the American Roentgen Ray Society (1904) show a normal chest and a tuberculous chest. Dr. Hulst attained these short exposures by substituting for the interruptor-coil a static machine made to his specifications, with 50 shellac and mica plates 28 inches in diameter, and 50 stationary glass plates 32 inches in diameter, a machine tested to perform at 7,000 r.p.m., but functioning at 1,800. His achievement was honored by the presidency of the American Roentgen Ray Society for 1905 and 1906.

This reporter has urged Mrs. Hulst, whose copper-plate handwriting betrays no inroads of age on either hand or brain, to write the history of Dr. Hulst and his times; it would be a precious source-book of one of the most productive periods in science. She replied, "I should like to do it, but feel incompetent to do it—I am too near. I have been thinking that you will want to know that Doctor Hulst said of his own work that he had always been a playboy and had never worked on medicine or the x-ray—and I think that is correct. He was not a plodder, and took endless pains because he was so interested."

From Mrs. Hulst, from Dr. Henry Duiker of Grand Rapids, and from the early publications of this Society, it is learned that Dr. Hulst was born in The Netherlands, in the province of Friesland, on June 25, 1859. He came to America in 1874, when his father, the Reverend L. J. Hulst, accepted a call to the church in Danforth, Illinois. He received his A.B. from Hope College, in Holland, Michigan; then, following in his father's footsteps, he entered the study of theology at Princeton. However, he soon realized that he desired more to be a physician, and the following year he entered the medical school of the University of Michigan, and received his M.D. in 1888.

Mrs. Hulst tells us, "He began his medical practice as an assistant physician at the State Asylum for the Insane in Traverse City, where he became interested in hypnotism as a therapeutic agent, and after visiting clinics in Amsterdam and Paris he applied it in his practice and wrote several notable papers on the subject." (E.g., "Gastrostomy in Hypnosis" *Medical Record*, 1897, *51*, 43.) Dr. Duiker states that "When patients began to ask to be hypnotized over the telephone, he decided that his notoriety in hypnotism would prevent his practicing medicine ethically, so he dropped the practice in hypnosis and became interested in x-ray."

Dr. Lewis Gregory Cole, who pays eloquent tribute in "Lung Dust Lesions Versus Tuberculosis" to Dr. Hulst's pioneer work in roentgen diagnosis of the chest, speaks on page 311 of that volume of asking a pathologist, a personal friend, for some lungs, and being told "You can't have those lungs. X-ray is only a *black art*, and anyone who has anything to do with it is a *charlatan*. For the sake of our family acquaintance I won't let you do this, and furthermore, I shall do anything I can to prevent you from getting them elsewhere." Dr. Hulst's work—or as he preferred to regard it, play—with roentgen rays advanced radiology so abundantly that it interests this writer to muse on whether, had Dr. Hulst stayed with hypnotism, so obviously useful a psychiatric tool would have gone as far into eclipse as it has.

The historian of the American Roentgen Ray Society has had close professional association with Dr. John R. Carty, Dr. Raymond W. Lewis, and Dr. William Snow, all three of whom have written ably on soft

tissue roentgenography; and he had gotten the idea that soft tissue roentgenography was rather a recent refinement. He finds that Dr. Hulst read a paper before the International Congress of Radiology in Amsterdam, published in the *New York Medical Journal* for January 22, 1908, volume 88, page 1224, under the title "Soft Tissue Roentgenography." He discusses contrast versus detail; and how does this sound for 1909? "The first period of roentgenology, the period of screaming contrasts, passes over into that of detail in soft tissues."

In 1913 and 1914, your reporter can testify first-hand, professors of anatomy were still holding students to know under which left ribs fore and aft the stomach was located. In 1905 Dr. Hulst reported, "A cursory examination of the 22 cases described discloses the fact that the x-ray picture of the normal stomach and colon, semidistended with milk and bismuth, is not what our text-books of anatomy would lead us to expect. . . . If the text-books are correct I have not yet succeeded in finding a normal stomach. Extended x-ray observations will probably change the current conception of this organ."

Dr. Hulst was thirty-six and a half years old when Röntgen discovered the x-ray. He was only fifty-four years old when severe diabetes forced his retirement from practice. His place among radiology's immortals was earned before 1914. A Grand Rapids colleague comments, "His situation is peculiar in that he has so long outlived his contact with his specialty. For that reason few of the radiologists in this area knew him or had even met him." Self-preservation turned his attention to diabetes, and in the words of Dr. Duiker, "insulin gave him a new lease on life. He began making exhaustive studies of diabetes on himself, making thousands of blood sugar readings and keeping detailed notes of all his findings. In spite of his severe diabetes, he lived to be nearly 90 and he died of arteriosclerosis, which led to a cerebral thrombosis." Although he was very early in radiology, and consequently incurred no little damage to his hands, he lost no fingers. This relatively good fortune may well be due to his early development of short exposures, at a time when his contemporaries were observing the tube with their hands as a test-object, during the course of long ones.

Dr. Duiker remarks, "Dr. Hulst had a scientific and a camera mind, and remembered when and where and on what page things were written. His knowledge of philosophy and theology was amazing. He kept up with newer developments in medicine, economics, art, and a variety of subjects. Mrs. Hulst also was well read and very intellectual. She is almost ninety years of age and very alert mentally. An evening spent with Dr. and Mrs. Hulst was a truly stimulating experience."

The American Roentgen Ray Society pays its tribute to one of the truly great builders of the foundation of radiology; and to the rare spirit who survives him "after fifty-nine years of rare companionship."

Ramsay Spillman, M.D.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Netherland Plaza Hotel, Cincinnati, Ohio, Oct. 4–7, 1949.

American Radium Society
Secretary, Dr. H. F. Hare, 605 Commonwealth Ave., Boston, Mass. Annual meeting: 1950, to be announced.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Cleveland Auditorium and Statler Hotel, Cleveland, Ohio, Dec. 4–9, 1949.

American College of Radiology
Executive Secretary, William C. Stronach, 20 N. Wacker Drive, Chicago 6. Annual meeting: 1950, to be announced.

Section on Radiology, American Medical Association
Secretary, Dr. U. V. Portmann, Cleveland Clinic, Cleveland, Ohio, Annual Meeting: 1950, to be announced.

Alabama Radiological Society
Secretary, Dr. W. W. Anderson, Tuscaloosa, Ala. Meets time and place Alabama State Medical Association.

Arizona Association of Pathologists and Radiologists
Secretary, Dr. R. Lee Foster, 507 Professional Bldg., Phoenix, Ariz. Two regular meetings a year. The annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Brooklyn Roentgen Ray Society
Secretary, Dr. Joseph Daversa, 603 Fourth Ave., Brooklyn, N. Y. Meets monthly fourth Tuesday, Oct. to April.

Buffalo Radiological Society
Secretary, Dr. Mario C. Gian, 610 Niagara St., Buffalo, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Roentgen Ray Society
Secretary, Dr. Dwight V. Needham, 608 E. Genesee St., Syracuse N. Y. Meets January, May, November.

Central Ohio Radiological Society
Secretary, Dr. Paul D. Meyer, Grant Hospital, Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, December, February, April, and June at Seneca Hotel, Columbus, Ohio.

Chicago Roentgen Society
Secretary, Dr. John H. Gilmore, 720 N. Michigan Ave., Chicago 11, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. Eugene L. Saenger, 735 Doctors Bldg., Cincinnati 2, Ohio. Meets last Monday of each month, September to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. J. R. Hannan, Cleveland Clinic, Cleveland 6, Ohio. Meetings at 6:30 P.M. on fourth Monday of each month from October to April.

Colorado Radiological Society
Secretary, Dr. Mark S. Donovan, 306 Majestic Bldg., Denver 2, Colo. Meets third Friday of each month at Department of Radiology, Colorado School of Medicine.

Connecticut Valley Radiologic Society
Secretary, Dr. E. W. Godfrey, 1676 Boulevard, West Hartford, Conn. Meets second Friday Oct. and April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, on third Monday, 7:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. W. G. Belanger, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434-30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

Florida Radiological Society
Secretary, Dr. F. K. Hurt, Riverside Hospital, Jacksonville, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

Georgia Radiological Society
Secretary, Dr. Robert Drane, DeRenne Apartments, Savannah, Ga. Meets in mid-winter and at annual meeting of Medical Association of Georgia in the spring.

Houston X-ray Club
Secretary, Dr. Curtis H. Burge, 3020 San Jacinto St., Houston 4, Texas. Meets fourth Monday each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. William M. Loehr, 712 Hume-Mansur Bldg., Indianapolis 4. Meets second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

Kansas Radiological Society
Secretary, Dr. Anthony F. Rossitto, Wichita Hospital, Wichita, Kan. Meets annually with State Medical Society.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Long Island Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Wybren Hiemstra, 1414 S. Hope St., Los Angeles 15, Calif. Meets second Wednesday each month at Los Angeles County Medical Assn. Building.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Louisville Radiological Society
Secretary, Dr. E. L. Pirkey, Louisville General Hospital, Louisville 2, Ky. Meets monthly on second Friday at Louisville General Hospital.

* Secretaries of societies are requested to send timely information promptly to the Editor.

Michigan Association of Roentgenologists
Secretary, Dr. R. D. McDuff, 220 Genesee Bank Bldg., Flint 3, Mich.

Milwaukee Roentgen Ray Society
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. Chauncey N. Borman, 802 Medical Arts Bldg., Minneapolis 2, Minn. Two meetings yearly, one at time of Minnesota State Medical Association the other in the fall.

Nebraska Radiological Society
Secretary, Dr. Ralph C. Moore, Nebraska Methodist Hospital, Omaha 3, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

New Hampshire Roentgen Ray Society
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Radiological Society
Secretary, Dr. J. E. Hemphill, 1420 E. Fifth St., Charlotte 4, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. C. O. Heilman, 807 Broadway, Fargo. Meetings held by announcement.

Northern California Radiological Club
Secretary, Dr. C. E. Grayson, Medico-Dental Bldg., Sacramento 14, Calif. Meets at dinner last Monday, every second month, except June, July and August.

Ohio State Radiological Society
Secretary, Dr. Carroll C. Dundon, 2065 Adelbert Road, Cleveland 6, Ohio.

Oklahoma State Radiological Society
Secretary, Dr. W. E. Brown, Tulsa, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. Boyd Isenhart, 214 Medical Dental Bldg., Portland 5, Oregon. Meets monthly 2nd Wednesday, 8:00 P.M., Library of University of Oregon Medical School.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. S. J. Hawley, 1320 Madison St., Seattle 4, Wash. Meets annually in May.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. J. M. Converse, 416 Pine St., Williamsport. Annual Meeting: May 20 and 21, 1949, Bedford Springs Hotel, Bedford, Pa.

Philadelphia Roentgen Ray Society
Secretary, Dr. Arthur Finkelstein, Graduate Hospital, 19th and Lombard St. Meets first Thursday each month October to May, at 8:00 P.M., in Thomson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. R. P. Meader, 4002 Jenkins Arcade Pittsburgh 22, Pa. Meets 6:30 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

Queens Roentgen Ray Society
Secretary, Dr. J. E. Goldstein, 88-29 163rd St., Jamaica 3, N. Y. Meets fourth Monday of each month except during the summer.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Harry A. Miller, 2452 Eutaw Place, Baltimore. Meets third Tuesday each month, September to May.

Radiological Section, Connecticut Medical Society
Secretary, Dr. Fred Zaff, 135 Whitney Ave., New Haven, Conn. Meets bimonthly on second Wednesday.

Radiological Section, District of Columbia Medical Society
Secretary, Dr. A. A. J. Den, 1801 K St., N. W., Washington, D. C. Meets Medical Society Auditorium, third Thursday, January, March, May, October at 8:00 P.M.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Radiological Society of New Jersey
Secretary, Dr. Raphael Pomeranz, 31 Lincoln Park, Newark, N. J. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Ralph E. Alexander, 101 Medical Arts Bldg. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. Maurice D. Frazer, 1037 Stuart Bldg., Lincoln, Nebr. Meets Shirley-Savoy Hotel, Denver, Colo. August 18, 19, 20, 1949.

St. Louis Society of Radiologists
Secretary, Dr. C. J. Nolan, 737 University Club Bldg., St. Louis 3, Mo. Meets fourth Wednesday each month, except June, July, August, and September.

San Diego Roentgen Society
Secretary, Dr. R. F. Niehaus, 1831 Fourth Ave., San Diego, Calif. Meets monthly, first Wednesday at dinner.

Section on Radiology, California Medical Association
Secretary, Dr. D. R. MacColl, 2007 Wilshire Blvd., Los Angeles 5, Calif.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. Harold L. Shinall, St. Joseph's Hospital, Bloomington, Ill.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas. Next meeting, Dallas, Texas, February 3 and 4, 1950.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May inclusive. Room 203, Service Memorial Institute, 426 N. Charter St., Madison.

Utah State Radiological Society
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake City 1, Utah. Meets third Wednesday in September, November, January, March and May.

Virginia Radiological Society
Secretary, Dr. P. B. Parsons, Norfolk General Hospital, Norfolk, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Homer V. Hartzell, 310 Stimson Bldg., Seattle 1, Wash. Meets fourth Monday each month, October through May, College Club, Seattle.

X-Ray Study Club of San Francisco
Secretary, Dr. W. F. Reynolds, University of California Hospital, San Francisco. Meets from January to July, 1949, at Lane Hall, Stanford University Hospital, and from July to December 1949, at San Francisco Hospital.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Mexico

Sociedad Mexicana de Radiologia y Fisioterapia
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Ordinary meeting, on the Thursday preceding the third Friday, October to May at 8:15 P.M.
Medical Members' meeting, on third Friday in each month at 5:00 P.M., 32 Welbeck St., London, W 1.

Faculty of Radiologists
Honorary Secretary, Dr. J. F. Bromley, 45, Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

Canadian Association of Radiologists
Honorary Secretary, Dr. E. M. Crawford, 1535 Sherbrooke St., West, Montreal 26, Que. Meetings January and June.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Francaise d'Electrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East, Montreal, P. Q.

Australian and New Zealand Association of Radiologists
Honorary Secretary, Dr. Alan R. Colwell, 135 Macquarie St., Sydney, N.S.W.
Honorary Secretaries, State Branches:
New South Wales, Dr. E. W. Frecker, 135 Macquarie St., Sydney.
Victoria, Dr. T. I. Tyrer, 3 Lockerbie Court, East St. Kilda.
Queensland, Dr. J. Adam, 131 Wickham Terrace, Brisbane.
South Australia, Dr. B. C. Smeaton, 178 North Terrace, Adelaide.
Western Australia, Dr. A. M. Nelson, 179-B St. Georges Terrace, Perth.
New Zealand, Dr. E. G. Lynch, 12 Bolton St., Wellington.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Sociedade Brasileira de Radiologia Medica
Secretary, Dr. Nicola Caminha, Av. Mem de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Andrelino Amaral, Av. Brigadeiro Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Tuesday at 9 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado, 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A Carrión," Villalta, 218, Lima.

Sociedad de Radiologica, Cancerologia y Fisica Medica del Uruguay
Secretary, Dr. Arias Bellini.

Continental Europe

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 111 Avenue des Alliés, Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

Ceskoslovenská společnost pro röntgenologii a radiologii v Praze
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Next meeting, Krakow, June 2 and 3, 1949.

Gdansk Section, Polish Society of Radiology
Secretary, Dr. A. Smigielska, Akademia Lekarska, Gdansk. Meets monthly last Sunday at 10.30, X-Ray Dept., Akademia Gdansk.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Meets monthly.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad.
USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Schweizerische Röntgen-Gesellschaft (Société Suisse de Radiologie)
President, Dr. H. E. Walther, Gloriastr. 14, Zürich, Switzerland.

Societa Italiana di Radiologia Medica
Secretary, Prof. Mario Ponzio, Ospedale Mauriziano Torino, Italy. Meets biannually.

PRELIMINARY PROGRAM

FIFTIETH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Fiftieth Annual Meeting of the American Roentgen Ray Society will be held at the Netherland Plaza Hotel, Cincinnati, Ohio, on October 4, 5, 6, and 7, 1949.

The Executive Council will meet at noon on Sunday, October 2.

The annual Golf Tournament for the Willis F. Manges Trophy will be held on Monday, October 3, at the Kenwood Country Club.

The Caldwell Lecture will be given on Wednesday evening, October 5, and the Annual Banquet will be held on Thursday evening, October 6.

The Instruction Courses will begin at 8:30 A.M., each day of the meeting, and elsewhere in this issue will be found the detailed plan of the courses.

The Scientific Program has been arranged as follows:

Tuesday, October 4, 1949

8:30 A.M. Instruction Courses.

10:00 A.M. Study of Scientific and Commercial Exhibits.

11:00 A.M. Call to Order, Fiftieth Annual Meeting, Lawrence Reynolds, M.D., President.

Presentation of Scientific Awards by the President.

Installation of the President-Elect, U. V. Portmann, M.D., Cleveland, Ohio, by President Reynolds and the Chairman of the Executive Council, Hugh F. Hare, M.D., Boston, Mass.

Address of Welcome: Raymond Walter, President of the University of Cincinnati.

Inaugural Address: President U. V. Portmann, M.D.

Paper No.

1. Study of Human Arteries by Different Radiographic Methods. Otto Glasser, Ph.D., Cleveland, Ohio.

2:00 P.M.

2. The Value of Tannic Acid and Post-evacuation Films. Arthur C. Christie, M.D., Fred O. Coe, M.D., Aubrey O. Hampton, M.D., and George M. Wyatt, M.D., Washington, D.C.

Discussion by Harry M. Weber, M.D., Rochester, Minn.

3. An Aid to the Diagnosis of Polypoid Lesions of the Colon. Robert M. Potter, M.D., Chicago, Ill. (by invitation).

Discussion by Paul C. Swenson, M.D., Philadelphia, Pa.

4. A Study of End-to-End Anastomosis of the Colon following Colonic Resection. Meyer Sharpe, M.D. (by invitation) and Ross Golden, M.D., New York, N.Y.

Discussion by James T. Case, M.D., Chicago, Ill.

5. Gastrointestinal Radiographic Observations of Patients treated by Vagotomy. C. A. Priviteri, M.D., Chamblee, Ga. (by invitation).

Discussion by Ross Golden, M.D., New York, N.Y.

6. Renal Cysts. Harold C. Ochsner, M.D., Indianapolis, Ind.

Discussion by H. Dabney Kerr, M.D., Iowa City, Ia.

4:30 P.M. Executive Business Session of the Society.

Wednesday, October 5, 1949

8:30 A.M. Instruction Courses.

10:00 A.M. Study of Scientific and Commercial Exhibits.

11:00 A.M.

7. Traumatic Diaphragmatic Hernia. B. Noland Carter, M.D., and Jerome Giuseffi, M.D., Cincinnati, Ohio (both by invitation).

Discussion by Charles M. Barrett, M.D. Cincinnati, Ohio (by invitation).

8. An Epidemic of Acute Granulomatous Pneumonitis. Benjamin Felson, M.D., George F. Jones, M.D., and Robert P. Ulrich, M.D., Cincinnati, Ohio (all by invitation).

Discussion by Albert B. Sabin, M.D., Cincinnati, Ohio.

9. Pulmonary Calcifications—Tuberculo-

sis? Histoplasmosis? Frederic N. Silverman, M.D., Cincinnati, Ohio (by invitation).

Discussion by W. Edward Chamberlain, M.D., Philadelphia, Pa.

2:00 P.M.

10. Alveolar Cell Tumors of the Lung. C. Allen Good, M.D., J. R. McDonald, M.D. (by invitation), and O. T. Clagett, M.D. (by invitation), Rochester, Minn.

Discussion by Donald B. Effler, M.D., Cleveland, Ohio (by invitation).

11. Mediastinal Emphysema. John A. Evans, M.D., and Ted R. Smalldon, M.D., New York, N.Y. (both by invitation).

Discussion by Leo G. Rigler, M.D., Minneapolis, Minn.

12. Unusual Lesions Involving the Vertebrae and Intervertebral Discs in Children. Eugene L. Saenger, M.D., Cincinnati, Ohio (by invitation).

Discussion by Ralph S. Bromer, M.D., Bryn Mawr, Pa.

13. Poisoning Due to Ingestion of Excess Vitamin A. John Caffey, M.D., New York, N.Y.

Discussion by Merrill C. Sosman, M.D., Boston, Mass.

14. Osseous and Articular Complications of Hypervitaminosis D. William R. Christensen, M.D., Charles Liebman, M.D. (both by invitation), and Merrill C. Sosman, M.D., Boston, Mass.

Discussion by John Caffey, M.D., New York, N.Y.

Wednesday Evening, October 5, 1949

Eight-Thirty O'Clock
The Caldwell Lecture
Stanley P. Reimann, M.D.

Director of the Research Institute, Lankenau Hospital, Philadelphia, Pennsylvania;
"The Differentiation of Cells and Tissues"
Introduction—Bernard P. Widmann, M.D., Philadelphia, Pennsylvania.

Thursday, October 6, 1949

8:30 A.M. Instruction Courses.

10:00 A.M. Study of Scientific and Commercial Exhibits.

11:00 A.M.

Symposium: Application of Radioactive Isotopes. Arranged by Western Reserve University, School of Medicine, Cleveland, Ohio.

15. Description of a Sr^{90} Beta Ray Applicator and Its Use on the Eye. Hymer L. Friedell, M.D., Charles I. Thomas, M.D., and Jack S. Krohmer, B.S., Cleveland, Ohio (all by invitation).

Discussion by Roscoe J. Kennedy, M.D., Cleveland, Ohio (by invitation).

16. Use of Radioactive Phosphorus in the Treatment of Carcinoma of the Breast with Widespread Metastases to Bone. Hymer L. Friedell, M.D., and John P. Storaasli, M.D., Cleveland, Ohio (both by invitation).

Discussion by T. Leucutia, M.D., Detroit, Mich.

17. I^{131} in the Diagnosis and Treatment of Hyperthyroidism. R. A. Shipley, M.D., John P. Storaasli, M.D., Hymer L. Friedell, M.D., and Albert Potts, M.D., Cleveland, Ohio (all by invitation).

Discussion by U. V. Portmann, M.D., Cleveland, Ohio.

2:00 P.M.

18. Clinical Isodose Curves. B. S. Wolf, M.D., and R. Loevinger, Ph.D., New York, N.Y. (both by invitation).

Discussion by William Harrris, M.D., New York, N.Y.

19. Design Characteristics for Standard Ionization Chambers up to 500 kv. From the National Bureau of Standards. R. J. Kennedy, Ph.D., and W. Snyder, B.S., Washington, D.C. (both by invitation).

Discussion by Lauriston S. Taylor, Ph.D., Washington, D.C.

20. Biological Effects of High Energy Roentgen Rays. Henry Quastler, M.D., Urbana, Ill. (by invitation).

Discussion by Kenneth E. Corrigan, Ph.D., Detroit, Mich.

21. Carcinoma of the Cervix Given Roentgen and Fractionated Radium Therapy Concurrently. Howard B. Hunt, M.D., and Robert M. Coleman, M.D., (by invitation), Omaha, Neb.

Discussion by Ralph M. Caulk, M.D., Washington, D.C.

22. Urethane in the Treatment of Multiple Myeloma. R. Wayne Rundles, M.D.

(by invitation) and Robert J. Reeves, M.D., Durham, N.C.

Discussion by T. Leucutia, M.D., Detroit, Mich.

4:30 P.M. Executive Business Session of the Society.

Thursday Evening. October 6, 1949

Seven-Thirty O'Clock
Annual Banquet

Friday, October 7, 1949

8:30 A.M. Instruction Courses.

10:00 A.M. Study of Scientific and Commercial Exhibits.

11:00 A.M.

Symposium: Combined Management of Hematologic and Allied Diseases from the Medical and Radiologic Viewpoints.

Arranged by Ohio State University, College of Medicine, Columbus, Ohio.

23. Leukemia, Polycythemia and Related Diseases. Claude-Starr Wright, M.D., Columbus, Ohio (by invitation).

Discussion by William E. Howes, M.D., Brooklyn, N.Y.

24. Hodgkin's Disease and Allied Disorders. Herman Hoster, M.D., Columbus, Ohio (by invitation).

Discussion by Maurice Lenz, M.D., New York, N.Y.

25. Diagnosis and Treatment of Lymphoblastoma and Leukemia from the Standpoint of the Radiologist. Joseph L. Morton, M.D., Columbus, Ohio (by invitation).

Discussion by Harold W. Jacox, M.D., New York, N.Y.

2:00 P.M.

26. Clinical and Technical Problems in Angiography. Russell H. Morgan, M.D., Baltimore, Md.

Discussion by Wendell G. Scott, M.D., St. Louis, Mo.

27. Coronary Angiography. James A. Helmworth, M.D., Johnson McGuire, M.D., and Benjamin Felson, M.D., Columbus, Ohio (all by invitation).

Discussion by J. Harold Katte, M.D., Columbus, Ohio (by invitation).

28. Angiographic Studies of the Pulmonary Artery. Joseph E. Miller, M.D., Dallas, Texas (by invitation).

Discussion by Fred J. Hodges, M.D., Ann Arbor, Mich.

29. The Angiocardiographic Diagnosis of Operability in Lung Cancer. Charles T. Dotter, M.D., Israel Steinberg, M.D., and Cranston W. Holman, M.D., New York, N.Y. (all by invitation).

Discussion by LeRoy Sante, M.D., St. Louis, Mo.

30. Cerebral Arteriography. C. Robert Hughes, M.D., Robert E. Wise, M.D., and John R. Hannan, M.D., Cleveland, Ohio (all by invitation).

Discussion by W. James Gardner, Cleveland, Ohio (by invitation).

AMERICAN ROENTGEN RAY SOCIETY

SECTION ON INSTRUCTION

HARRY M. WEBER, M.D., *Director*

Titles and Abstracts of Courses Offered
Fiftieth Annual Meeting
Netherland Plaza Hotel, Cincinnati, Ohio
October 4–7, 1949

PRESIDENT-ELECT Portmann, with the approval of the Executive Council, has directed that the Section on Instruction be continued for the annual meeting of 1949. His program has been arranged so that the Instruction Courses will be offered between the hours of 8:30 A.M. and 10:00 A.M. on Tuesday, Wednesday, Thursday and Friday. This year only one course will be offered in each of the eleven rooms assigned for this purpose. Thus a total of 90 minutes has been made available for each course which makes it possible for instructors to devote more time to discussion and to conference with those attending the course. No other official activity has been scheduled for this time.

The Section on Instruction presents for 1949:

1. A Symposium on Radioactive Isotopes (Courses 101, 102, 103), with a faculty of three instructors and covering three periods.

2. Two Special Courses, one on the Preparation and Presentation of Medical Papers (Course S-1), and one on Radiation Dosimetry (Course S-2), with a faculty of two instructors and covering two periods.

3. Three Sequential Courses, two in Diagnostic Roentgenology (Courses B and C), and one in Therapeutic Radiology (Course A), with a faculty of three instructors and covering six periods.

4. Twelve single period courses in Therapeutic Radiology (Courses 201–212), with a faculty of twelve instructors and covering twelve periods.

5. Twenty-one single period courses in Diagnostic Roentgenology (Courses 301–321), with a faculty of sixteen instructors and covering twenty-one periods.

GENERAL INFORMATION

The Faculty

Vincent W. Archer, M.D., Professor of Roentgenology and Chairman School of Roentgenology, Department of Medicine, University of Virginia, Charlottesville, Virginia.

W. E. Baensch, M.D., Professor of Roentgenology, Goergetown University Hospital, Washington, D.C.

Carl B. Braestrup, Senior Physicist, Department of Hospitals, New York City; Associate Professor of Radiology, New York University and Columbia University, New York, New York.

Ralph M. Caulk, M.D., Attending Radiologist and Medical Director of the Tumor Clinic, Garfield Memorial Hospital, Washington, D.C.

W. Edward Chamberlain, M.D., Professor of Radiology, Temple University Medical School and Head of the Department of Radiology, Temple University Hospital, Philadelphia, Pennsylvania.

Richard H. Chamberlain, M.D., Assistant Professor of Radiology, University of Pennsylvania, School of Medicine, Philadelphia, Pennsylvania.

Arthur E. Childe, M.D., Radiologist, Children's Hospital of Winnipeg; Associate Radiologist, Winnipeg General Hospital, Winnipeg, Manitoba, Canada.

K. E. Corrigan, Ph.D., Director, Research Division, Harper Hospital, Detroit, Michigan.

Edwin C. Ernst, M.D., Director, Radiological Department, Barnard Free Skin and Cancer Hospital, Saint Louis, Missouri.

Robert E. Fricke, M.D., F.A.C.R., Associate Professor of Radiology, Mayo Foundation, University of Minnesota; Consultant in Section on Therapeutic Radiology, Mayo Clinic, Rochester, Minnesota.

C. Allen Good, M.D., M.S. in Radiology. Assistant Professor of Radiology, Mayo Foundation, University of Minnesota; Radiologist, Mayo Clinic, Rochester, Minnesota.

H. F. Hare, M.D. Radiologist, Lahey Clinic, Boston, Massachusetts.

Richard M. Hewitt, M.A., M.D., Assistant Professor of Medical Literature, Mayo Foundation, University of Minnesota; Editor and Head of Division of Publications, Mayo Clinic, Rochester, Minnesota.

Fred Jenner Hodges, M.D., Professor of Roentgenology, University of Michigan, Ann Arbor, Michigan.

John F. Holt, M.D., Associate Professor of Roentgenology, University of Michigan, Ann Arbor, Michigan.

Howard B. Hunt, M.D., Professor and Chairman of the Department of Radiology, University of Nebraska, College of Medicine, Omaha, Nebraska.

Harold W. Jacox, M.D., Professor of Radiology, College of Physicians and Surgeons, Columbia University; Chief of Radiotherapy Division of Radiological Service, Presbyterian Hospital, New York, New York.

B. R. Kirklin, M.D., F.A.C.P., F.A.C.R., Professor of Radiology, Mayo Foundation, University of Minnesota; Radiologist and Chairman of Sections on Radiology, Mayo Clinic, Rochester, Minnesota.

Isadore Lampe, M.D., Associate Professor of Roentgenology, University of Michigan, Ann Arbor, Michigan.

Maurice Lenz, M.D., Professor of Clinical Radiology, Columbia University, New York; Consultant Radiotherapist to the Manhattan Eye, Ear and Throat Hospital, Montefiore Hospital and Presbyterian Hospital, New York, New York.

Robert S. MacIntyre, M.D., Assistant Professor of Roentgenology, University of Michigan, Ann Arbor, Michigan.

A. S. Macmillan, M.D., Radiologist, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.

Charles L. Martin, M.D., Professor of Radiology, Southwestern Medical College, Dallas, Texas.

Henry G. Moehring, M.D. Radiologist, Duluth Clinic, Duluth, Minnesota; Formerly Director of School of Roentgenology at Army School of Roentgenology, Memphis, Tennessee.

Edward B. D. Neuhauser, M.D., Associate in Radiology, Harvard Medical School, Boston, Massachusetts; Radiologist to The Infant's and to The Children's Hospitals of Boston.

H. M. Parker, M.A., Ph.D., Manager of Health Instrument Divisions, General Electric Company Nucleonics Department, Richland, Washington.

Lester W. Paul, M.D., Professor of Radiology, University of Wisconsin, Madison, Wisconsin.

Carleton B. Peirce, A. B., M.Sc., M.D., F.A.C.P., Radiologist-in-Chief, Royal Victoria Hospital; Chairman, Department of Radiology, Faculty of Medicine, McGill University, Montreal, Quebec, Canada.

Harold O. Peterson, M.D., Clinical Associate Professor of Radiology, University of Minnesota, Minneapolis, Minnesota; Radiologist to Miller Hospital and Children's Hospital, Saint Paul, Minnesota.

Everett L. Pirkey, M.D., Associate Professor of Radiology, University of Louisville School of Medicine; Director, Department of Radiology, Louisville General Hospital, Louisville, Kentucky.

Ernst A. Pohle, M.D., Ph.D., F.A.C.R., Professor of Radiology; Chairman, Department of Radiology, University of Wisconsin Medical School, Madison, Wisconsin.

Leo G. Rigler, M.D., Professor of Radiology, University of Minnesota, Minneapolis, Minnesota.

Wendell G. Scott, M.D., Associate Professor of Clinical Radiology, Washington University School of Medicine; Associate Director, Mallinckrodt Institute of Radiology, Saint Louis, Missouri.

C. A. Stevenson, M.D., Head of the Department of Radiology, Scott and White Clinic, Temple, Texas.

Marcy L. Sussman, M.S., M.D., Radiologist to the Mt. Sinai Hospital, New York; Clinical Professor of Radiology, Columbia University, New York; Phoenix, Arizona.

Bernard P. Widmann, M.D., Chief Radiologist, Philadelphia General Hospital; Chairman of the Graduate School in Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.

Conference Periods

Tuesday, Wednesday, Thursday and Friday mornings

Single Periods.....8:30–10:00 A.M.

Location

All courses will be given in Parlors A, B, C, D, E, F, G, H, I, J, and L, located on the Fourth Floor of the Netherland Plaza Hotel. Full information may be obtained at the general registration desk which will be located near by.

Code

The instruction periods will be designated with the following code:

T, Tuesday.....8:30–10:00 A.M.
W, Wednesday.....8:30–10:00 A.M.
Th, Thursday.....8:30–10:00 A.M.
F, Friday.....8:30–10:00 A.M.

How to Register and Obtain Tickets for the Instruction Courses

Admission to the Instruction Courses will be by ticket only.

Following the list of titles and abstracts is a general order sheet. First, second and third choices for each period should be selected carefully and indicated on the order. The rooms in which the courses will be given are moderately large ones, but it will still be necessary to place a limitation on the number of persons who will be able to attend individual courses. It is estimated that the number who can attend will range between 50 and 100 persons. If the directions given on the order sheet are followed explicitly, errors in completing reservations will be minimized.

Persons requesting registration in one or more of the Sequential (continuous) Courses (A, B, C) should indicate second and third choices of single-period courses as substitutes for each period—this is to avoid disappointment should the Sequential Courses be filled when the order is received.

It is possible for one to attend only four periods of instruction, so the condensed schedule given on the last pages of this program should be consulted with care when registering and ordering tickets.

Reservations will be made in the order in which the order forms are received. Those who are not members of the American Roentgen Ray Society will be charged the nominal fee of $1.50 per period of instruction, or a maximal fee of $5.00 for four periods. Full-time graduate students in Radiology will be admitted without fee, but they are required to make application for the courses they wish to attend.

Previous to September 24, 1949, the order forms should be sent to the Director, Dr. Harry M. Weber, Mayo Clinic, Rochester, Minnesota. After September 24, the orders should be sent directly to Dr. Harry M. Weber, Netherland Plaza Hotel, Cincinnati, Ohio.

In case the courses are not filled by the time of the meeting, tickets will be available at the registration desk on Sunday, October 2, and thereafter during the meeting.

Holders of Tickets

Those who do not have the proper ticket for the course they wish to attend will not be permitted to enter the room. Pages will be in attendance in each of the conference rooms to collect the tickets.

DESCRIPTION OF COURSES

I. The Symposium on Radioactive Isotopes

COURSE 101

Room L **Period: F**

CARL B. BRAESTRUP, Ph.D., New York, N. Y.

Planning the Hospital Radioisotope Facilities

Setting up a radioisotope program involves the appointment of a supervising committee in the hospital, obtaining suitable laboratory space, securing competent personnel to do the actual work, pro-

curing the necessary equipment and instruments for safe handling and proper measurement of isotopes, and filing the necessary forms with the Atomic Energy Commission.

All these requirements will be discussed and detailed consideration will be given to instrumentation and radiologic safety.

COURSE 102

Room L **Period: T**

K. E. CORRIGAN, Ph.D.,
Detroit, Mich.

Radioactive Isotopes, Their Production and Properties

A brief summary of the historical background with the derivation and precise meaning of modern isotope terminology will introduce the course. The useful methods of production, including low and high voltage accelerators, the cyclotron and the nuclear reaction pile with the type of isotopes produced by each, will be discussed. Transmutation reactions and the preparation and purification of useful isotopes will be considered. The properties which make certain radioisotopes useful in medical diagnosis and therapy will be considered in detail. The limitations on the choice of an isotope, particularly with respect to the radiological toxicity of the long-lived isotopes, will be presented. The course will include the practical classification of the medically useful isotopes as they are known at the present time, both with respect to their use in elemental or simple inorganic forms, and to the present status and future possibilities of more complex organic compounds.

COURSE 103

Room L **Period: Th**

HOWARD B. HUNT, M.D.,
Omaha, Nebraska

Practical Utilization of Radioisotopes in Radiotherapy

A practical presentation of the principles of protection, radioassay, instrumentation, clinical indications, dosimetry and therapeutic results essential to utilization of radioiodine in thyroid disease and radiophosphorus in polycythemia and leukemia.

II. Sequential Courses

COURSE A
(2 periods)

Room A **Periods: Th; F**

CARLETON B. PEIRCE, M.D.,
Montreal, Quebec

Radiation Therapy of Malignant Neoplasms Involving the Brain and Central Nervous System

A brief survey of the types of
(a) Primary neoplasms
(b) Common systemic neoplasms
(c) Common metastatic neoplasms affecting the brain, brain stem and cord will be followed by a discussion of the characteristics of each which affect the selection of cases for irradiation.

This discussion will be further elaborated with a consideration of such methods of irradiation, in which the major emphasis will be given to the procedures used in the Royal Victoria Hospital and the Montreal Neurological Institute.

Certain special problems, such as medulloblastoma, the systemic neoplasms, such as the lymphogranuloma group and the common metastatic carcinoma will be included if time permits.

COURSE B
(2 periods)

Room G **Periods: Th; F**

HAROLD O. PETERSON, M.D.,
St. Paul, Minn.

The Roentgenologic Examination of the Urinary Tract

The material for this course has been taken from an active urologic service at the Miller Hospital in St. Paul, Minnesota, supplemented by cases from University of Minnesota Hospitals. The course will deal largely with intravenous or excretion urography. It will be divided into two parts. In the first period the routine procedures will be thoroughly discussed, including the preparation of the patient, use of cathartic drugs, pitressin, dehydration, types of contrast media used, compression, time interval for films, position of patient, etc. Also presented in this first period will be the excretory urographic appearance of the normal urinary tract, and the variations from the normal which are not considered to have pathologic significance. In the second period the lesions of the urinary tract will be presented together with some of the important congenital anomalies of the urinary tract which can be recognized by intravenous urography.

Emphasis will be placed on practical procedures and on the urologic abnormalities more commonly encountered, rather than on the theoretical and the unusual. Original roentgenograms will be used for illustration with the use of a special projector.

COURSE C
(2 periods)

Room I **Periods: Th; F**

MARCY L. SUSSMAN, M.D.,
Phoenix, Arizona

Angiocardiographic Technique and Interpretation

This course will present a discussion of angiocardiography including the technique, the applications and the limitations. Particular attention will be paid to the results obtained in congenital heart disease, in cor pulmonale, in mitral disease, and in lesions of the lung and mediastinum. More briefly, techniques such as cardiac catheterization will be considered in their relation to problems in which angiocardiography is useful.

III. Special Courses

COURSE S-1

Room J **Period: Th**

RICHARD M. HEWITT, M.D.,
Rochester, Minn.

Writing of Medical Papers

Some tricks of the trade which assist the journeyman medical writer in construction of his whole essay, his paragraphs, his sentences and his tabular and textual stereoptican slides.

COURSE S-2

Room L **Period: W**

H. M. PARKER, M.A., Ph.D.,
Richland, Wash.

Radiation Dosimetry

1. Roentgen rays and gamma rays:
   Advantages of an ionization method.
   Interpretation of the original definition of the "roentgen".
   Energy absorption and the Bragg-Gray Principle.
   Interpretation of the current definition of the "roentgen".
   The gamma-roentgen.
   Complexities with multi-million volt radiation.
2. Other radiations:
   Extension of the roentgen to particulate radiations.
   Gray's Energy Unit.
   The "rep".
   Applications to radioisotope therapy and neutron therapy.
   Other proposals for physical dose units.
3. Factors of biological effectiveness:
   Relative biological effectiveness.
   The "rem".
   Scale of relation between "rep" and "rem".
   Severe limitations of the "rem".

IV. Therapeutic Radiology

COURSE 201

Room D **Period: T**

RALPH M. CAULK, M.D.,
Washington, D. C.

Transvaginal Roentgen Therapy in Carcinoma of the Cervix Uteri

Roentgen therapy by the transvaginal method has been used in the treatment of over 450 cases since October, 1946, in the Department of Roentgen Therapy at the Garfield Memorial Hospital.

Experience indicates that this is one of the most efficacious methods by which the primary tumor can be modified and controlled.

Apparatus, technique and dosage will be demonstrated, both by lantern slides and on a wax model.

The place of this method in the treatment schema will be discussed.

Results in terms of five year survivals and the complications incurred, will be fully emphasized.

COURSE 202

Room G **Period: T**

W. EDWARD CHAMBERLAIN, M.D.,
Philadelphia, Pa.

Some Harmful Effects of Irradiation and and Their Avoidance

Irradiation is a two-edged sword. Correctly applied in a properly selected case its potentialities for good may be boundless: its potentialities for harm must, however, be borne in mind at all times. When

irradiation is applied in the treatment of a benign condition, resultant harm to normal structures will not be acceptable.

Cases will be presented in which harm resulted from (*a*) a single exposure, (*b*) a series of relatively minute exposures. There will be emphasis upon the significance of the interval between small exposures and evidence will be produced to indicate that great caution is necessary in the use of irradiation for such conditions as acne, epidermophytosis, psoriasis, and the common wart.

COURSE 203

Room J **Period: T**

RICHARD H. CHAMBERLAIN, M.D.,
Philadelphia, Pa.

Low Voltage-Short Distance Therapy

In the treatment of superficial and accessible lesions, roentgenologic apparatus which produces long wavelength roentgen rays and can be operated at short distances may offer appreciable advantages. Precise control of depth dosage, easy and quick application, and good uniformity of field coverage is possible. Beryllium window tubes now enlarge the scope of radiation quality obtainable.

In clinical practice, new judgments of dosage are necessary. Excellent results are obtained in accessible epitheliomas, superficial metastases, hemangiomas, benign conditions of the cornea and sclera. The avoidance of unfavorable deep radiation effects and the optimal cosmetic results are particularly gratifying.

COURSE 204

Room C **Period: F**

E. C. ERNST, M.D.,
St. Louis, Mo.

Practical Concepts of Radiation Treatment of Carcinoma of the Cervix Uteri

SYNOPSIS

(a) *Clinical Management and Preliminary Considerations*
(b) *Indications for Roentgen Therapy:*
  1. Indirect irradiation of the pelvis.
  2. Direct (intravaginal) roentgen-ray applications.
(c) *Essential Tumor Dose Measurement Factors*
(d) *Indications for Radium Therapy:*
  1. Evaluation of the various methods and the intracervical applicators.
  2. Essential minimum requirements for obtaining the ideal uniform distribution of radium radiations.
(e) *Prognostic Factors:*
  1. Tumor grading.
  2. Stage of the disease.
  3. Initial response to preliminary roentgen irradiation.
(f) *Final Discussion Period:*
  Case presentations.
  2. Questions (15 minutes).

ABSTRACT

The practical irradiation management of carcinoma of the cervix will be discussed both from the standpoint of the institutional tumor clinic and the private office procedure. Although realizing that the radiation treatment standards continue to remain somewhat in a state of flux and that individualization in the application of roentgen rays and radium is a most essential consideration, nevertheless certain fundamental concepts in our routine procedures are most helpful in the management of cancer of the cervix. These and many other practical therapeutic considerations, including external roentgen therapy, intracavity radium and roentgen methods of treatment and the dosage measurement problems, will be discussed and illustrated.

COURSE 205

Room J **Period: F**

R. E. FRICKE, M.D.,
Rochester, Minn.

Beta Irradiation in Diseases of the Eye

The elementary physics of beta particles will be discussed. Work in various centers has shown promising healing in the treatment of early corneal ulcers, leukoma, benign papillomas, vernal conjunctivitis, recurring pterygium and other inflammatory dyscrasias. Early results are good. Beta particles are furnished by applicators containing radon, radium D or radioactive strontium. Dosage problems will be considered, and apparatus designed and used at the Mayo Clinic for radium D therapy will be shown.

COURSE 206

Room C **Period: W**

HUGH F. HARE, M.D.,
Boston, Mass.

Symposium—Cancer of the Thyroid

Thyroid cancers arise in pre-existing tumors. The clinical evaluation of an individual case does not ex-

clude cancer. Single thyroid nodules are malignant in 10 per cent of the cases coming to operation. We believe radical surgery followed by roentgen therapy is the treatment of choice with the exception of malignant fetal adenomata, which we do not believe are clinically malignant.

A roentgen-ray tumor dose of 4,800 to 6,000 roentgens is necessary to control the growth of papillary cyst adenocarcinoma, papillary adenocarcinoma, and small cell carcinoma. Giant cell carcinoma does not respond to doses of roentgen rays as outlined here. Radium is the useful adjunct in residual and infiltrative tumor, especially adenocarcinoma. An attempt should be made to give at least 6,000 gamma roentgens to the periphery of the tumor.

Ten to twenty year follow-up studies show that good results have been obtained in the treatment of (*a*) fetal adenomata with malignant transformation; (*b*) papillary cyst adenocarcinoma; (*c*) papillary adenocarcinoma. Poor results are obtained in the treatment of alveolar adenocarcinoma and giant cell carcinoma. The failure of treatment of the small cell type of thyroid cancer is usually a result of its widespread infiltration at the time the patient presents himself for treatment.

Associated hyperthyroidism is rare in thyroid cancer, occurring approximately in 8 per cent. Radioactive iodine as a method of treatment will be discussed.

COURSE 207

Room C **Period: Th**

HAROLD W. JACOX, M.D.,
New York, N. Y.

Radiation Treatment of Tumors of the Kidney and Adrenal Glands

Since these tumors are primarily of surgical importance an attempt will be made to correlate the urologic and radiologic viewpoints.

Highlights of the clinical aspects of interest to radiologists will be given. Technical procedures rather than statistical results will be emphasized. Some of the difficult problems will be illustrated by case reports.

COURSE 208

Room J **Period: W**

ISADORE LAMPE, M.D.,
Ann Arbor, Mich.

Radiation Therapy of Selected Miscellaneous Non-Malignant Conditions

A discussion of clinical indications, value and techniques of radiation therapy in the following conditions:

Simple inflammations and infections
Acute parotitis
Acute postpartum mastitis
Tuberculous lymphadenitis
Rheumatoid spondylitis
Myasthenia gravis
"The thymus".

COURSE 209

Room C **Period: T**

MAURICE LENZ, M.D.,
New York, N. Y.

Roentgen Therapy of Cancer of the Breast

Experience with roentgen therapy of mammary cancer at Presbyterian Hospital, New York, between 1923 and 1944 will be discussed. Five year arrests by this treatment has required tumor doses of 6,000 to 8,000 roentgens in three to four months. This has been practical only in tumors limited to the breast and the axilla. Smaller dosage has resulted in growth restraint of shorter duration.

COURSE 210

Room D **Period: F**

CHARLES L. MARTIN, M.D.,
Dallas, Texas

Treatment of Cancer of the Face, Mouth and Metastatic Cervical Lymph Nodes with Irradiation

All carcinomas of the face and mouth, regardless of their size, are treated with either roentgen therapy or low intensity radium needles. In many instances electrosurgery is used as a preliminary procedure to reduce the tumor to a flat plane or to remove involved cartilage. Metastatic lymph nodes in the neck are treated by a combination of roentgen therapy and low intensity radium needles. Details of technique, illustrative cases and statistical results will be presented with the aid of lantern slides.

COURSE 211

Room A **Period: T**

ERNST A. POHLE, M.D.,
Madison, Wisconsin

Radiation Therapy of Vascular Nevi

Vascular nevi of the skin may be divided into five groups: (1) capillary hemangiomas (port wine

stain); (2) hemangioma simplex (strawberry mark); (3) cavernous hemangioma; (4) spider-like capillary telangiectasia; (5) lymphangioma. Treatment recommended by various authors consists of (*a*) injection with a sclerosing solution (quinine-urethane); (*b*) CO_2 snow; (*c*) radium; (*d*) roentgen rays; (*e*) desiccation. In the Department of Radiology at the University of Wisconsin we have used radium in the majority of our cases; roentgen rays occasionally, especially for large lesions, and desiccation for the spider-like nevus. The techniques of treatment and the results obtained will be discussed in detail.

COURSE 212

Room D **Period: W**

BERNARD P. WIDMANN, M.D.,
Philadelphia, Pa.

Bronchogenic Carcinoma—Radiation Treatment

Bronchogenic carcinoma is curable only by radical surgery. The incidence of operability is relatively slight. The preponderance of cases must be regarded for palliative irradiation or no treatment. This discussion will be an attempt to classify the results of roentgen treatment of bronchogenic carcinoma at the Philadelphia General Hospital during the past twenty years. The beneficial effects with special reference to the longevity cycle have been tabulated in a series of more than 400 cases. A manifold variety of techniques, as well as the skin and physical tolerance for varying rates and total doses, has been analyzed according to the results obtained. The beneficial effects of irradiation are very definite for about one-third of the treated cases. The longevity cycle is not appreciably altered even for the patients who are improved in health and strength and clinically very much benefited. This experience, however, emphasizes the importance and necessity of cultivating more interest and enthusiasm for a greater effort to carry out irradiation more systematically and vigorously for bronchogenic carcinoma.

V. Diagnostic Roentgenology

COURSE 301

Room B **Period: T**

V. W. ARCHER, M.D.,
University, Virginia

Diagnosis of Bone Tumors

On the basis of experience with proved cases of bone tumor the difficulty of precise roentgenologic diagnosis of many of them will be discussed. Misleading original pathologic reports and misleading clinical symptoms often add to the diagnostic difficulties. The role of blood chemistry studies, biopsy, roentgenologic data, clinical history and examinations will be stressed. Lesions simulating malignant tumors of bone will be shown and discussed.

COURSE 302

Room B **Period: W**

W. E. BAENSCH, M.D.,
Washington, D. C.

The Mucosal Pattern of the Stomach and Duodenum

A technique of demonstrating the pattern of the mucous membrane of the stomach and duodenum will be described. The physiologic basis for the development of the normal pattern will be discussed and compared with the changes in the pattern developed in various gastric and duodenal diseases. Emphasis will be placed on the practical aspects of roentgenologic observations of this kind.

COURSE 303

Room E **Period: T**

ARTHUR E. CHILDE, M.D.,
Winnipeg, Manitoba

The Manifestations of Intracranial Disease in Plain Roentgenograms of the Skull

This course will deal with the evidence of intracranial disease revealed in roentgenograms of the skull made without the use of contrast media. Such manifestations of intracranial disease will be discussed as intracranial calcification, changes in the bony structure of the calvarium, changes in the sella turcica, calcification of the pineal gland and significant displacement of its shadow, changes in the vascular markings of the calvarium. Lesions of the scalp and of the skull which sometimes simulate the manifestations of intracranial disease will also be given due consideration.

COURSE 304

Room E **Period: W**

ARTHUR E. CHILDE, M.D.,
Winnipeg, Manitoba

The Normal Encephalogram and Ventriculogram Congenital Abnormalities of the Brain

The roentgen technique of cerebral pneumography will be discussed and the importance of a few simple

manipulations of the head during this procedure will be explained. This will be followed by a review of the normal anatomy of the ventricular system, basal cisterns and cortical markings. The pneumographic features of various congenital abnormalities will be shown.

COURSE 305

Room E **Period: Th**

ARTHUR E. CHILDE, M.D.,
Winnipeg, Manitoba

The Pneumographic Diagnosis of Expanding, Contracting and Atrophic Intracranial Lesions

The deformities produced by various types of expanding intracranial lesions will be shown. Encephalography is often used to determine the cause of epileptic seizures in patients who do not suffer from brain tumors, and some examples of atrophic and contracting intracranial lesions will also be illustrated.

COURSE 306

Room F **Period: W**

C. ALLEN GOOD, M.D.,
Rochester, Minn.

The Roentgenologic Diagnosis of Surgical Lesions of the Mediastinum

With recent advances in thoracic surgery has come an increasing need for more accurate diagnosis of mediastinal lesions. This responsibility rests largely on the roentgenologist.

This course will include a discussion of the criteria for roentgenologic diagnosis of the lesions of the mediastinum which are generally considered to require surgical treatment. The lesions will be discussed in groups according to their origin in the anterior, middle or posterior mediastinum. Lesions of cardiac or of pulmonary origin will not be included except as they enter the differential diagnosis.

COURSE 307

Room E **Period: F**

FRED JENNER HODGES, M.D.,
Ann Arbor, Mich.

Intracranial Angiography

The indications for this type of examination will be outlined and the required technical procedures will be discussed. Examples of angiographic results in the examination of various patients typifying the major forms of intracranial abnormality which can be detected by this method will be presented.

Although this is a very highly specialized form of roentgen diagnosis with a relatively narrow field of application, the degree of diagnostic accuracy which it brings to neurosurgical problems is not to be overlooked. Even though a radiologist does not employ it himself he should be fully aware of its potentialities.

COURSE 308

Room B **Period: Th**

JOHN F. HOLT, M.D.,
Ann Arbor, Mich.

The Diagnosis of Benign and Malignant Tumors of Bone

A discussion of the virtues and limitations of roentgenology in the evaluation of destructive and proliferative lesions in bone due to neoplasm and allied diseases. Using illustrative roentgenograms, emphasis will be placed on significant generalities rather than minor details of individual lesions with the idea of clarifying the true position of the roentgen method in this important but somewhat controversial field of diagnosis.

COURSE 309

Room G **Period: W**

B. R. KIRKLIN, M.D.,
Rochester, Minn.

Cholecystographic Technique

The importance of meticulous technique in cholecystography will be discussed.

COURSE 310

Room F **Period: F**

ROBERT S. MacINTYRE, M.D.,
Ann Arbor, Mich.

Pulmonary Tuberculosis

Roentgenologic methods are well established in the detection and treatment of pulmonary tuberculosis. The uses and limitations of these methods will be discussed. Special attention will be given to the importance of childhood tuberculosis and of minimal tuberculous lesions in the adult. The basis for a practical approach in the handling of any given

patient will be emphasized. Selected roentgenograms will be used as illustrative material.

COURSE 311

Room H **Period: T**

A. S. MACMILLAN, M.D.,
Boston, Mass.

Roentgen Examination of the Sinuses

1. Roentgen examination of the sinuses is a very important part of the diagnosis of pathology in these locations. The rhinologist is dependent upon a correct interpretation of the sinus roentgenograms. The findings should be reported in terms of pathology, and not in the indefinite roentgenological terms so often used. In this presentation, the technique of examination and interpretation will be considered as fully as time will allow.

COURSE 312

Room H **Period: W**

A. S. MACMILLAN, M.D.,
Boston, Mass.

Roentgen Examination of the Mastoids

2. The use of chemotherapy and antibiotics has greatly changed the course of infection in the mastoid. Pediatricians and general medical practitioners sometimes feel competent to treat this condition, but too often the treatment is inadequate. The roentgenologic examination of the mastoid is the only dependable source of information. This should be recognized because mastoiditis is still prevalent. Acute and chronic infections of the mastoid and the complications will be discussed.

COURSE 313

Room H **Period: Th**

A. S. MACMILLAN, M.D.,
Boston, Mass.

3. *The film reading session* will consider conditions met in a hospital devoted to treatment of the sinuses, mastoids and abnormalities of the esophagus. Rare and unusual conditions are often emphasized in a presentation such as this, with failure to give adequate consideration to the more common types of pathology met with every day. This discussion will deal with the more commonly encountered abnormalities in which roentgenologic assistance is sought.

The new projector developed by Doctors Dillon and Murphy which makes it possible to show four original roentgenograms of the sinuses at one time will be used.

COURSE 314

Room F **Period: Th**

HENRY G. MOEHRING, M.D.,
Duluth, Minn.

Bronchographic Technique and Interpretation

A discussion of the anatomy of the bronchial tree and its nomenclature will introduce considerations of the clinical importance of the bronchopulmonary segments, several techniques for bronchography, and the chief indications for bronchography.

COURSE 315

Room I **Period: T**

EDWARD B. D. NEUHAUSER, M.D.,
Boston, Mass.

The Roentgen Diagnosis of Congenital Cardiovascular Abnormalities Amenable to Surgery

Many forms of congenital malformation of the heart and great vessels, long considered of academic interest only, are now surgically correctible. Recent advances in roentgen diagnosis and in surgical techniques are leading to the recognition of abnormalities which have been overlooked in the past, and have led to the more accurate diganosis of many malformations whose major features have long been known.

The roentgen features of patent ductus arteriosus, coarctation of the aorta, stenosis of the pulmonary artery, tetralogy of Fallot, Corvisart's complex, double aortic arch, vascular rings constricting the trachea and esophagus, and anomalies of the right subclavian artery and of the innominate vein should be familiar to all roentgenologists. The diagnostic criteria of these malformations will be discussed.

COURSE 316

Room A **Period: W**

EDWARD B. D. NEUHAUSER, M.D.,
Boston, Mass.

Certain Aspects of the Gastrointestinal Tract in Infants and Young Children

In this course, no effort will be made to cover all the possible abnormalities and diseases that are seen

or which reflect themselves in abnormalities of the alimentary tract.

Particular attention will be given to the technique of examination of the esophagus and gastrointestinal tract in infants and in young children and also to the technique of examination of the colon in infants.

Particular attention will be devoted to congenital anomalies and resulting diseases of the esophagus, certain unusual forms of herniation of the bowel into the thorax, and considerable attention will be given to the problems of duplication of the alimentary tract.

Malrotation of the bowel, congenital stenosis and atresia and study of the patient with imperforate anus will be discussed.

Several other anomalies and acquired lesions will be discussed briefly.

COURSE 317

Room D **Period: Th**

LESTER W. PAUL, M.D.,
Madison, Wisconsin

The Roentgenology of the Rheumatic Diseases

This course will include a discussion of the role of roentgen examination in the diagnosis of the various diseases of the joints and periarticular structures. A classification of arthritis suitable for both roentgen and clinical use will be given. The pathologic basis for the changes visualized by roentgen examination will be covered briefly. Major emphasis in the course will be placed upon the correlation of the roentgenologic and clinical manifestations of the various types of joint disease, and upon the roentgenologic signs by which these types are distinguished from each other, and by which progression from early to late stages is followed.

COURSE 318

Room B **Period: F**

EVERETT L. PIRKEY, M.D.,
Louisville, Kentucky

Differential Diagnosis of Lesions in and About the Fundus of the Stomach

I. Review of principal anatomical relations about the stomach.
II. Evolution of methods for diagnosis of gastric lesions.
III. Description of our new method.
IV. Indications for use.
V. Typical cases demonstrating its value.

COURSE 319

Room F **Period: T**

LEO G. RIGLER, M.D.,
Minneapolis, Minn.

Roentgen Manifestations of Bronchogenic Tumors

The roentgen findings obtained with various types of bronchogenic tumors will be discussed with particular reference to the following:

1. The possibilities and limitations of roentgen diagnosis of bronchogenic tumors.
2. The roentgen delineation of the tumor mass.
3. Inflammatory changes associated with bronchogenic tumors.
4. The roentgen evidences of bronchial obstruction such as emphysema, atelectasis, and secondary inflammatory changes.
5. The demonstration of the bronchial tumor by bronchography and planigraphy.
6. The special findings in bronchial adenoma.

Diagrams and reproductions of roentgenograms will be demonstrated to illustrate these various findings.

COURSE 320

Room I **Period: W**

WENDELL G. SCOTT, M.D.,
St. Louis, Mo.

Angiographic Procedures in the Diagnosis of Congenital Heart Disease

This course will include a discussion of the development and description of equipment for the rapid automatic serialization of roentgen exposures synchronized with a Potter-Bucky grid with special emphasis on the use of the Rapidograph with roll film.

The techniques of injection for both cardiovascular angiography and aortography will be discussed.

Indications and contraindications for the use of angiographic procedures as related to the diagnosis of congenital heart disease will be mentioned. A series of lantern slides will be used to demonstrate the roentgenographic findings in various types of congenital heart disease.

COURSE 321

Room H **Period: F**

C. A. STEVENSON, M.D.,
Temple, Texas

The Conduct of the Roentgenologic Examination of the Colon

The roentgenologist is responsible for the detection of any organic lesion of the colon above the level of proctoscopic reach. In order to accept this responsibility, many exacting criteria must be fulfilled. The subjects to be discussed are (1) detailed consideration of the methods of preparing the patient for the intestinal examination; (2) comparison of various suspensions of barium; (2) mechanical aids used during the roentgenoscopic examination; (4) roentgenoscopic procedure, and (5) roentgenographic technique.

Studies on the nature of pseudo-polyps and their differentiation from true polyps will be discussed. Emphasis will be given to the method of producing superior double contrast films.

CONDENSED SCHEDULE OF COURSES ON TUESDAY, OCT. 4

Code: T	8:30 A.M.–10:00 A.M.		Code: T
Therapeutic Radiology and Radiation Physics		**Diagnostic Roentgenology**	
201—Caulk	Transvaginal Roentgen Therapy in Carcinoma of the Cervix Uteri	**301—Archer**	Diagnosis of Bone Tumors
202—Chamberlain, W. E.	Harmful effects of Irradiation and Their Avoidance	**303—Childe**	Manifestations of Intracranial Disease in Plain Roentgenograms of the Skull
203—Chamberlain, R. H.	Low Voltage-Short Distance Therapy	**311—Macmillan**	Roentgen Examination of the Sinuses
102—Corrigan	Radioactive Isotopes, their Production and Properties	**315—Neuhauser**	Roentgen Diagnosis of Congenital Abnormalities Amenable to Surgery
209—Lenz	Roentgen Therapy of Cancer of the Breast	**319—Rigler**	Roentgen Manifestations of Bronchogenic Tumors
211—Pohle	Radiation Therapy of Vascular Nevi		

CONDENSED SCHEDULE OF COURSES ON WEDNESDAY, OCT. 5

Code: W	8:30 A.M.–10:00 A.M.		Code: W
Therapeutic Radiology and Radiation Physics		**Diagnostic Roentgenology**	
206—Hare	Cancer of the Thyroid	**302—Baensch**	Mucosal Pattern of the Stomach and Duodenum
208—Lampe	Radiation Therapy of Selected Miscellaneous Non-Malignant Conditions	**304—Childe**	Normal Encephalogram and Ventriculogram; Congenital Abnormalities of the Brain
S-2—Parker	Radiation Dosimetry	**306—Good**	Roentgenologic Diagnosis of Surgical Lesions of the Mediastinum
212—Widmann	Bronchogenic Carcinoma	**309—Kirklin**	Cholecystographic Technique
		312—Macmillan	Roentgen Examination of the Mastoids
		316—Neuhauser	Gastrointestinal Tract in Infants and Young Children
		320—Scott	Angiographic Procedures in the Diagnosis of Congenital Heart Disease

CONDENSED SCHEDULE OF COURSES ON THURSDAY, OCT. 6

Code: Th — **8:30 A.M.–10:00 A.M.** — **Code: Th**

Therapeutic Radiology and Radiation Physics		Diagnostic Roentgenology	
103—Hunt	Practical Utilization of Radioisotopes in Radiotherapy	305—Childe	Pneumographic Diagnosis of Expanding, Contracting and Atrophic Intracranial Lesions
207—Jacox	Radiation Treatment of Tumors of the Kidney and Adrenal Glands	308—Holt	The Diagnosis of Benign and Malignant Tumors of Bone
A—Peirce	Radiation Therapy of Malignant Neoplasms Involving the Brain and Central Nervous System	313—Macmillan	Film Reading Session
		314—Moehring	Bronchographic Technique and Interpretation
S-1—Hewitt	Writing of Medical Papers	B—Peterson	Roentgenologic Examination of the Urinary Tract
		C—Sussman	Angiocardiographic Technique and Interpretation
		317—Paul	Roentgenology of the Rheumatic Diseases

CONDENSED SCHEDULE OF COURSES ON FRIDAY, OCT. 7

Code: F — **8:30 A.M.–10:00 A.M.** — **Code: F**

Therapeutic Radiology and Radiation Physics		Diagnostic Roentgenology	
101—Braestrup	Planning the Hospital Radioisotope Facilities	307—Hodges	Intracranial Angiography
204—Ernst	Practical Concepts of Radiation Treatment of Carcinoma of the Cervix Uteri	310—MacIntyre	Pulmonary Tuberculosis
205—Fricke	Beta Irradiation in Diseases of the Eye	B—Peterson	Roentgenologic Examination of the Urinary Tract
210—Martin	Treatment of Cancer of the Face, Mouth and Metastatic Cervical Lymph Nodes with Irradiation	318—Pirkey	Differential Diagnosis of Lesions in and about the Fundus of the Stomach
A—Peirce	Radiation Therapy of Malignant Neoplasms Involving the Brain and Central Nervous System	321—Stevenson	Conduct of Roentgenologic Examination of the Colon
		C—Sussman	Angiocardiographic Technique and Interpretation

Vol. 62, No. 1

ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

Department Editor: George M. Wyatt, M.D., 1835 Eye St., N.W., Washington 6, D. C.

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ROENTGEN DIAGNOSIS

HEAD

White, J. C., Liu, Ching Tung, and Mixter, W. J. Focal epilepsy; epilepsy secondary to intracranial tumors. *New England J. Med.*, June 24, 1948, *238*, 891–899.

Of 1,130 patients admitted to the Massachusetts General Hospital with all varieties of epilepsy in the decade 1935 through 1944, 240, or 21 per cent, had seizures arising from a focal area that could be localized by clinical examination, roentgen studies, or electroencephalography. The seizures in the 240 patients were usually, but not always, of the jacksonian type. Of the 240 cases, 160 were caused by intracranial tumors, and 80 by trauma or infection. All of the cases were surgically explored.

The 160 cases caused by tumor constituted 25 per cent of a group of 641 brain tumors operated on during the same decade. In the latter group, the incidence of seizures varied with the location of the tumor, being highest for tumors close to the motorsensory areas of the cerebral cortex, and lowest for infratentorial neoplasms. Tumors situated deeply in the basal ganglia or third ventricle rarely gave rise to seizures.

Age of onset is an important factor to consider in determining the etiology of seizures. When epilepsy begins in adult life with a previous history of cranial trauma, abscess, or encephalitis, the most probable diagnosis is brain tumor.

Grouping of tumors by cytologic origin shows that the greatest incidence of seizures occurs in the most slowly growing tumors: astrocytoma (50 per cent), meningioma (35 per cent), oligodendroglioma (37 per cent), and hemangioma (33 per cent). Glioblastoma multiforme and metastatic carcinoma have lesser epileptogenic tendencies (22 per cent and 14 per cent respectively). However, in those cases of glioblastoma where seizures did appear, they were the first evidence of the disease in 75 per cent. Pituitary tumors produce seizures only after escaping the sella to invade the cerebral hemispheres.

Postoperatively, epilepsy accompanying brain tumor is often ameliorated and sometimes completely relieved. Epilepsy not present before operation occasionally appears postoperatively. —*Henry P. Brean, M.D.*

Nathanson, Louis, and Losner, Samuel. Ossification of auricles of external ears associated with acromegaly. *Radiology*, Jan., 1947, *48*, 66–68.

The authors report their findings in a patient with classical acromegaly in whom marked calcification or ossification was noted in the lobes of both ears. The ears were roentgenographed because of their appearance and rigidity. Both auricles revealed involvement of the entire cartilage. Because of the trabeculated appearance of the calcified structure, the authors thought they were dealing with bone rather than ordinarily deposited calcium.

Reviewing the literature, it was learned that cartilage frequently undergoes changes of a proliferative and generative nature in acromegaly. Similar calcification and ossification may take place in the ribs, intervertebral discs, trachea and larynx.

Pathologically, the process is characterized by an increase in cartilaginous cells and intercellular substance which leads to a distention of the transitional zone between bone and cartilage. Followed by degeneration with liquefaction and formation of vacuoles in the deficient cartilaginous tissue, the process eventuates in ossification which may take place with or without previous calcification.—*Philip J. Hodes, M.D.*

Childe, Arthur E., and Young, Arthur W. Pneumographic diagnosis of intraventricular epidermoid. *Radiology*, Jan., 1947, *48*, 56–60.

Intracranial epidermoids are comparatively rare. The reported incidence is variable but probably less than 1 per cent. Intraventricular epidermoids are even less common but have an unmistakable pneumographic appearance. The authors report a case in which the diagnosis was made preoperatively.

Plain roentgenograms of the skull were noncontributory. The encephalograms suggested a block above the fourth ventricle. Ventriculography showed dilatation of the posterior portions of both lateral ventricles and dilatation of both temporal horns. The anterior portions of both lateral ventricles were enlarged upward and reached to within 1.5 cm. of the inner table of the skull. These portions of the ventricles were almost filled by soft tissue masses through and around which multiple furrows containing gas were visible. The tumor masses did not extend into the posterior horns. The third ventricle was poorly visualized but contained oxygen in its inferior portion. At operation a typical

epidermoid was removed from the lateral and third ventricles.—*John R. Hannan, M.D.*

King, Joseph E. J. Diploic epidermoid and extra-dural pneumatocele: cranial defects and deformity. *Ann. Surg.*, May, 1948, *127*, 925–952.

The author presents two unusual types of cranial defects: (1) diploic epidermoids and (2) extradural pneumatocele of spontaneous origin. He supports his statement that preoperative diagnosis can always be made in these cases with clear-cut descriptions of the characteristic roentgen findings in each group.

The diploic epidermoids produce two types of skull defects: (1) Infrequently the tumor will be completely extracranial with erosion of the outer table only producing a doughy tumor which does not pulsate or show an impulse on coughing. (2) In the majority of these neoplasms arising from congenital epidermal rests in the diploe there is destruction of the inner table. The margin of the defect is scalloped, dense and clear cut. Bony hiatuses may be observed in the outer table. It is believed that the density of bone of this margin is due to the compression of the bone by the slowly growing tumor. There is no invasion of the bone. Lesions range in size from 1.5 cm. to 7.5 cm. in diameter or more. Rate of growth is very slow. The treatment of choice is block excision for complete removal unless the underlying dura contains a vital structure such as the lateral sinus when one must be content with curettage and sclerosing solution.

The accumulation of air between the dura and skull without perforation of the skull is a rare condition. The author reports 2 cases. On the skull films the cranial defect gives the impression of "coral rock" with well-defined, finely scalloped margins. Grossly there is irregular erosion of bone which is associated with sharp projections, stalactitic in character. There is no clear explanation for this appearance of bone other than the pressure of the air. For treatment the involved portion of bone should be removed, and the air fistula should be located and sealed off.—*T. D. Allison, M.D., and C. L. Hinkel, M.D.*

Van Metre, Thomas E., Jr. Malignant tumors of the nasopharynx. *Bull. Johns Hopkins Hosp.*, Jan., 1948, *82*, 42–55.

A review of 46 cases of malignant tumor of the nasopharynx is given. A rather adequate table giving the clinical picture of all the cases is presented. Other tables showing the type of tumors found, the age of patients involved, classification of the type of first symptoms, interval between first symptoms and hospital admission, and incidence of types of signs and symptoms on admission are also presented. In all cases a visible growth was present in the nasopharynx. The main signs and symptoms were those of pressure, obstruction, and destruction caused by the growth. Roentgenograms of the skull were obtained in 21 of the 46 patients. These were of much value diagnostically, however, in that definite erosion of the base of the skull was shown in 12 cases and soft tissue shadow of tumor displacing the nasopharyngeal air column was demonstrated in 7 instances. In only 7 cases did roentgenograms fail to show evidence of tumor and this was probably due to the fact that anteroposterior, lateral and stereoscopic films of the skull were not taken routinely. Therapy and mortality values are not analyzed, but a table of the significant autopsy findings of 5 cases is included.—*Jerome J. Wiesner, M.D.*

NECK AND CHEST

McClendon, J. F., Foster, Wm. C., and Cavett, J. W. An attempt to explain the anomalous action of Lugol's solution in exophthalmic goiter. *Endocrinology*, March, 1948, *42*, 168–173.

Using chicks as the experimental animal, one group was fed thiouracil, one group Lugol's solution and one group used as controls. The thyroid and pituitary glands were analyzed for protein-bound iodine. Thiouracil markedly increased the weight of the thyroid glands and reduced the protein-bound iodine to 10 per cent of normal. The protein-bound iodine of the pituitary glands was reduced to 85 per cent of its normal value.

Lugol's solution increased the protein-bound medicine of the thyroid 133 per cent and of the pituitary 48 per cent. It seems probable that local action of this protein-bound iodine in the pituitary might inhibit the release of thyrotropin and explain the anomalous action of Lugol's solution in exophthalmic goiter.—*John W. Karr, M.D.*

Wyman, Alvin C. Primary atypical pneumonia; roentgenographic course, complications,

recovery rate, and end results. *Dis. of Chest*, July–Aug., 1948, *14*, 568–579.

This is a statistical analysis of 855 cases of primary atypical pneumonia of unknown etiology in males, ranging in age from seventeen to thirty-five, at a naval recruit training center. The location of the pneumonia by lobe involvement was correlated with complications such as pleurisy or effusion, reinfection, recurrence, clearing time, and conditions of the lungs at the conclusion of the disease. Seventy-five per cent of the cases involved one or both lower lobes, with almost equal incidence in the other three lobes including multilobar involvement. The roentgenographic clearing time averaged 15.2 days without correlation between lobe involved and recovery rate in uncomplicated cases. About four additional days were required for clearing if pleurisy or effusion complicated the disease. Rare complications include atelectasis, pneumothorax, subcutaneous emphysema and bronchiectasis. Complete recovery was noted in 91 per cent, while 7 per cent showed residual pleural thickening or parenchymal fibrosis. Four cases of lower lobe bronchiectasis were proved by bronchography after reaching the chronic stage, to exclude the possibility of "pseudo-bronchiectasis."—*Fred R. Gilmore, M.D.*

Morgan, Russell H. The roentgenologic diagnosis of tuberculosis. *Bull. Johns Hopkins Hosp.*, March, 1948, *82*, 411–413.

The author states that two comprehensive investigations within recent years have shown that mass roentgenographic methods are as efficient as regular 14 by 17 inch films in the early detection of tuberculosis. In the ensuing discussion it was revealed that as many as 1,000 films can be read adequately in one hour. There is a subjective error in reading these films, however, in that a reader in reading a set of films for the second time may have a discrepancy of 15 per cent between the two readings—the same as the discrepancy between five different readers reading films simultaneously. Distance from the films is a factor in the ability to see the lesions. Where there was a difference of opinion in reading films among several readers, the majority rule was abided by. These roentgen-ray procedures are on sound ground in diagnosing tuberculosis, but the evaluation of the activity of the lesion is more difficult and the margin of error varies accordingly.—*Jerome J. Wiesner, M.D.*

Lincoln, Edith M., Stone, Samuel, and Hoffman, Olga R. The treatment of miliary tuberculosis with promizole. *Bull. Johns Hopkins Hosp.*, Jan., 1948, *82*, 56–75.

In a period of over seventeen years prior to this study 102 cases of miliary tuberculosis in children with roentgen evidence of mottling of the lungs were seen. All of these terminated fatally. In this study 11 cases of miliary tuberculosis which showed mottling of the lungs on the roentgenogram were treated with promizole. Dosage and toxic effects are discussed. Six case histories are given. Of these 11 children, 5 consecutive cases were apparently treated adequately with promizole and of these 2 are dead but 3 are alive thirty to thirty-three months after promizole was first given. Roentgenograms in these all showed recession of the miliary lesions. Also reported is a case of pulmonary miliary tuberculosis which developed in an adolescent girl with chronic hematogenous tuberculosis. Roentgenograms showed complete clearing of the miliary lesions after nine months of promizole therapy. Although promizole acts very slowly in children, it does seem to have a favorable action in suitable cases of miliary tuberculosis.—*Jerome J. Wiesner, M.D.*

Livingston, Samuel. Sedimentation rate in asthma in children. *Bull. Johns Hopkins Hosp.*, March, 1948, *82*, 385–388.

In a previous publication the author reported the results of treatment of a group of 34 asthmatic children by irradiation of the lymphoid tissue of the nasopharynx with radon. This group included patients with both intrinsic and extrinsic asthma and all had suffered frequent asthmatic attacks for at least two years prior to treatment. In this study the author reports the erythrocyte sedimentation rate in 33 of the 34 patients before and after treatment with radon. Of 22 patients who responded to the radon treatment (moderately to completely relieved of asthmatic attacks) 21 had a high sedimentation rate before treatment and in all but one of these the sedimentation rate returned to normal after radon therapy was completed. Of the 11 patients who failed to respond to radon treatment only one had an increased sedimentation rate before treatment. It was suggested, therefore, that the sedimentation rate determination is a good test in predicting which children with asthma will be benefited by irradiation and to differentiate between infectious and

non-infectious (allergic) asthma.—*Jerome J. Wiesner, M.D.*

Lenègre, J., Kilaidonis, P., and de Brux, J. Les calcifications de l'aorte ascendante. (Calcifications of the ascending aorta.) *Arch. mal du coeur.* May–June, 1948, pp. 193–210.

Thirty such cases as observed roentgenologically are reported. A routine fluoroscopic examination of the chest is superior to teleroentgenography. Use of more penetrating rays (under 4 to 5 ma.) and a thorough search of the aortic region (in the anterior and oblique, especially anterior left oblique, positions) with a well diaphragmed beam is stressed.

The average age incidence was sixty-one years. Twenty out of the 30 cases had proved syphilis. Of these 20 cases 11 did not show a positive serology at the stage of aortic calcification. The syphilitic calcifications are at the proximal part of the aorta, at the origin of the arch. If found at the horizontal portion they could always be traced to the ascending part of the aorta with a continuous borderline of more than a few centimeters, whereas atheromatous calcifications have a distal topography (usually on the horizontal part of aorta) with a scale-like opacity on the inferior left border of the aortic arch.—*J. N. Sarian, M.D.*

Meʟamed, Abraham, and Walker, Lynn J. Dissecting (intramural) pharyngo-esophageal diverticulum. *Radiology*, Dec., 1947, *49*, 712–716.

The authors present a case report of a dissecting (intramural) pharyngo-esophageal diverticulum of the pulsion type in a fifteen year old boy. The symptoms were present for one month prior to the examination and a surgical excision was performed nine months later.

The roentgen signs of this type of diverticulum are described as follows: (1) opacification and emptying of the diverticulum during examination in the upright position; (2) an apparent elevation or "lifting" of the diverticulum during deglutition, due to expulsion of its contents by the surrounding and contracting esophageal musculature; (3) origin in the midline posteriorly; (4) obscuration or "disappearance" of the opacified diverticulum in the lateral view, due to superimposition of the opaque shadows of the diverticulum and esophagus proper.—*Norman Heilbrun, M.D.*

ABDOMEN

Brummer, Pekka, and Bundul, Alfred. On the effect of some common gastric drugs on the motility of the stomach. *Acta med. Scandinav.*, June, 1948, *130*, 559–574.

The authors carried out roentgen examinations to determine the effect of the usual gastric drugs on the motility of the stomach. The drugs used were atropine, sodium barbital, papaverine, hydrochloric acid, and five different antacids. Atropine decreased and sodium barbital increased the gastric peristalsis. Papaverine did not affect gastric motility but decreased duodenal motility and tone. Hydrochloric acid, likewise, had no distinct effect on the gastric motility, while it increased the duodenal peristalsis, causing in particular retroperistalsis. Among the antacids, sodium bicarbonate very clearly increased the gastric peristalsis, which was also true, although in a less marked degree, of the other antacids used; the magnesium salts furthermore acted on the duodenal peristalsis. The authors believe that benefit from the drugs under observation is due to their effect on gastric and duodenal motility. This was further corroborated by therapeutic experiments in which sodium bicarbonate, which had the most distinct effect on the gastric motility, relieved gastric distress of patients distinctly better than other antacids which had equal or greater neutralizing powers. It cannot be said with certainty why one patient prefers one drug and another patient will prefer a different drug. However, the authors feel that further studies in motility will be more revealing than studies of secretion in determining the laws governing these therapeutic effects.—*Charles M. Nice, Jr., M.D.*

Ferguson, Ira A. Prolapse of the gastric mucosa. *Ann. Surg.*, May, 1948, *127*, 879–886.

The author states that prolapse of gastric mucosa through the pylorus is a distinct clinical entity with an incidence greater than that of gastric ulcer. In a review of 297 gastrointestinal examinations this condition was observed in 23 cases, or an incidence of 7.7 per cent. It is seen in males in a ratio of 5:1 and chiefly in the fourth decade although age range was from twenty to eighty years.

Although the etiology is unknown several interesting theories are discussed. One of these is based on low grade inflammation of the mucosa

by chronic irritation with resultant hypertrophy. Another cites the normal mobility of the gastric mucosa and believes that neurogenic factors may be sufficient to cause the prolapse without pre-existing disease.

The pathological changes are redundancy of the gastric mucosa and increased mobility in the prepyloric area. The portion of the mucosa which prolapses is often hyperemic. Ulceration and polyp formation of this mucosa have been reported. As the symptomalology is variable, an exact diagnosis rests on the roentgenographic findings which are distinctive. The author quotes from Pendergrass and Andrews for the roentgen features and includes the following: (1) the prolapsing gastric mucosa produces a central filling defect in the duodenal cap with a thin shadow of barium around the defect; (2) the size of the defect depends on the degree of the prolapse and the motor activity of the stomach and is usually not seen in the erect position; (3) the diagnosis can be easily missed by fluoroscopic examination; (4) there is no disturbance in the passing of the peristaltic waves and the duodenal bulb is not irritable; (5) there is usually a six hour residue; (6) there is a variable defect in the prepyloric region of the stomach when prolapsed mucosa extends through the pyloric ring although the redundant gastric rugae can usually be traced from the antral canal to the base of the duodenal bulb.

The treatment in most cases can be dietary management and antispasmodics but surgical intervention is required in the more advanced cases.—*T. D. Allison, M.D., and C. L. Hinkel, M.D.*

KIRSNER, JOSEPH B., PALMER, WALTER L., MAIMON, S. N., and RICHETTS, W. E. Clinical course of chronic nonspecific ulcerative colitis. *J.A.M.A.*, July 10, 1948, *137*, 922–928.

Literature pertaining to nonspecific ulcerative colitis continues to emphasize widely varying concepts as to pathogenesis and treatment. Few observations are recorded as to the natural history of the disease. For the purpose of inquiring into the natural history of this condition 100 out of a total of 250 patients were chosen because of completeness of data, length of period of observation and accurate information as to present status.

Nonspecific ulcerative colitis is a chronic disease whose course is characterized by exacerbations and remissions. This morbid process may possibly extend by retrograde spread from anus to terminal ileum. It was, however, surprising to note how frequently the initial involvement seemed to be the maximum involvement. Evidence of improvement could usually be obtained by proctoscopic examination.

The roentgen findings did not, as a whole, correlate with the clinical severity of the disease or with the duration of symptoms. By roentgen examination complete involvement of the colon was observed in 10 patients whose illness had existed less than one year, whereas a normal colon was demonstrated in 2 persons more than ten years after the initial appearance of their symptoms. The roentgen report of the colon was normal in 24 of 89 patients studied in this series.

Obviously the cause of nonspecific ulcerative colitis is obscure. Precipitating factors seem predominantly to be associated with emotional stress, acute infection of the respiratory tract and physical fatigue. In this series the number of complications were high. Complications can be divided into two groups: (1) those secondary to loss of nutrition such as cachexia, anemia, fatty degeneration of the liver; and (2) those local in nature such as perforation, abscess, fistula, stricture and neoplasm. Peritonitis secondary to bowel perforation, malnutrition and thrombosis accounted for the majority of deaths.

Lasting cure is rare following medical management comprising 6 per cent in this series. Forty-seven per cent of the patients were, however, maintained in a satisfactory state of health by medical regimen. Surgery restored 14 of 19 patients to satisfactory health. There were 5 deaths among the surgical cases. Ileostomy resulted in no cure. Six of the 9 patients who died under medical supervision died before 1942. The effect of more modern modalities of therapy (nutritional, antibiotic and psychiatric) on the course of this disease remain to be ascertained.—*William E. Howes, M.D.*

PRESENT, ARTHUR J., and GEYMAN, MILTON J. Diffuse calcification of the pancreas. *Radiology*, Jan., 1947, *48*, 29–32.

Present and Geyman report 2 cases of diffuse calcification of the pancreas. They state that the literature contains only 17 cases of pancreatic calcification, other than stones. One of their cases occurred in a white male, aged twenty-three, who, for a period of three years, suffered recurrent attacks of epigastric pain with nause, avom-

iting, and diarrhea. There was a preceding, questionable diagnosis of duodenal ulcer. The urine contained acetone and diacetic acid. Excretory urogram was normal, as was a cholecystogram. At operation, when the distal two-thirds of the pancreas was removed, only small patches of normal pancreatic tissue remained, the major portion of the pancreas being replaced by dense hyaline connective tissue containing numerous cysts and abscesses with multiple calcifications.

The second case was not proved by operation. The chief symptom was vague epigastric pain and the pancreas presented numerous calcifications. In discussing the differential roentgen diagnosis, distribution of the calcifications throughout the usual position of the pancreas is stressed. The authors state that pancreatic calculi are larger and usually confined to the head of the pancreas. Biliary and urinary calculi, calcified nodes, calcification of the costal cartilages, calcifications in the liver and spleen and adrenal calcifications are to be considered in the differential diagnosis. The use of air in the stomach may be helpful in localizing the calcifications.—*Robert C. Pendergrass, M.D.*

GYNECOLOGY AND OBSTETRICS

Howson, John Y. Pelvic cancer delay; organization and observations of the Philadelphia committee for the study of pelvic cancer. *Am. J. Obst. & Gynec.*, March, 1948, *55*, 538–540.

A committee of gynecologists has been formed in Philadelphia to study all pelvic cancer cases in Philadelphia with special reference to the sequence of events occurring between the onset of the patient's first symptoms, a definite diagnosis and adequate treatment. The facts obtained from this study are to be used to inform the medical profession of their importance in diminishing the delay in diagnosis of pelvic cancer. The committee meets monthly. Patients investigated during the preceding month are divided into those revealing no delay and those with an apparent delay. Those with an apparent delay are then divided into 3 groups: (1) delay due to the patient, (2) delay due to the physician, and (3) delay due to both. Physicians connected with the patients were invited to the meetings to discuss the cases—otherwise information obtained from the patient alone may be misleading. This also serves another purpose of education of the physician.

Between Nov. 1, 1945 and Jan. 1, 1947 some 455 living patients with pelvic cancer were investigated. Of this number there was no delay in 31.8 per cent and a delay of one or more months from the first symptoms until a correct diagnosis was made and adequate treatment instigated in the remaining 68.1 per cent. Of the cases showing a delay the patient was responsible in 63.6 per cent and the physician in 24.5 per cent. Patient and physician were dilatory in 12.0 per cent. In 82.3 per cent of the cases one physician was responsible for the delay, in 15 per cent two physicians were involved, and in 2.6 per cent three physicians were responsible. It is to be noted that in 113 cases showing physician delay a total of 136 doctors were responsible. These figures demonstrate that the physician plays an active part in the problem of delay in early diagnosis.—*Wendell C. Hall, M.D.*

Scheffey, Lewis C., Rakoff, A. E., and Hoffman, Jacob. An evaluation of the vaginal smear method for the diagnosis of uterine cancer. *Am. J. Obst. & Gynec.*, March, 1948, *55*, 453–460.

The authors have had four years' experience covering approximately 5,000 cases with the Papanicolaou technique of diagnosing uterine cancer from the cytology of vaginal smears. Vaginal smears were taken on all patients admitted to the gynecological wards, care being exercised not to take smears within twenty-four hours of a vaginal douche or other procedures which might interfere with the collection of material or staining reaction. The smears were all taken by one trained technician. The technique was that described by Papanicolaou and Traut. The smear from each case was given a number and reported so that the reader of the slide did not know the patient or clinical history. On the other hand, the pathologic diagnosis of removed tissue was submitted by the pathologist without any knowledge on his part of the findings of the cytologist. The study was continued until 500 consecutive cases had been completed.

The vaginal cytology smear is not intended to replace biopsy of the cervix or diagnostic curettage of the fundus, and even in most competent hands some cases of carcinoma will be missed by the vaginal smear method and there will be some false positives. The larger the number of samplings taken from a patient, the more accurate the results, but in the present series only one smear was taken from each patient for practical purposes. This is probably responsible for

the high percentage of positive cases missed in the present series, and since completion of the study the authors now get repeated smears on cases in which any bizarre cells are seen. Unquestionably the experience of the microscopist is of great importance in the success of the vaginal smear method. There is also a personal factor involved since some technicians have an excess of false positives whereas others have an excess of false negatives in their results.

The present study indicates the general value of the vaginal smear technique for routine study of gynecologic patients and the authors feel that this technique may well take its place as a routine public health diagnostic measure. The study emphasizes the importance of fully investigating every case presenting cells suggestive of malignancy, since the percentage of false positives is not high.—*Wendell C. Hall, M.D.*

SKELETAL SYSTEM

OLDFELT, C. O. Renal osteodystrophy; report of a case. I. Clinical aspect. *Acta med. Scandinav.*, May, 1948, *130*, 489–499.

The author presents the clinical aspects of a case of renal osteodystrophy in an adult woman who was thirty-three years of age when first seen. There were multiple areas of rarefaction in the bones of the pelvis, some of the long bones, phalanges and vertebrae. There was a strong tendency to calcification in the soft tissues of the abdominal wall, as well as in the arteries of the extremities. The blood calcium was normal, the secretion of calcium in the urine was not increased, and a marked renal insufficiency, with increased non-protein nitrogen and increased blood phosphorus, was present the whole time she was under observation. A history of urinary tract infections, normal blood pressure and urinary findings suggestive of contracted kidney suggested a diagnosis of chronic pyelonephritis. Primary hyperparathyroidism seemed unlikely, as the serum calcium and the secretion of calcium in the urine registered normal values. However, on clinical data it was possible that the case could have been one of primary hyperparathyroidism that came under observation only after advanced renal insufficiency had developed.—*Charles M. Nice, Jr., M.D.*

ENELL, HERBERT. Renal osteodystrophy. II. Pathological-anatomical account. *Acta med. Scandinav.*, May, 1948, *130*, 500–504.

This author gives the findings of the postmortem examination in the same case discussed in part I. There was a fairly homogeneous enlargement of the parathyroid glands, and since there was no pronounced adenoma formation the picture conforms most closely with a diffuse parathyroideal hyperplasia of the secondary type. No postmortem examination of the skull (and therefore of the hypophysis) was made. The kidneys were small and shrivelled, weighing 100 grams, macroscopically presenting the picture of severe nephrosclerosis and small parenchymal cysts. The microscopic picture revealed arteriosclerosis, while the somewhat larger vessels were relatively thin-walled and but slightly sclerotic. In the renal vessels of the size of art. arciformes there were extensive intimal calcifications of the Jores' type. There were also abundant calcifications in other organs of the body, including the endocardium, pleura, mesentery, splenic, femoral, uterine and hemorrhoidal vessels and the skin. The vertebrae presented a microscopic picture of diffuse disintegration of bone, with no signs of formation of new bone.

The pathologist agrees with the clinician that the condition was primarily of renal origin.—*Charles M. Nice, Jr., M.D.*

STEINBROCKER, OTTO, SPITZER, NORMAN, and FRIEDMAN, HAROLD H. The shoulder-hand syndrome. *Ann. Int. Med.*, July, 1948, *29*, 22–49.

This condition consists of a peculiar combination of painful shoulder disability with homolateral pain and swelling of the hand in otherwise healthy adults. The emphasis on the unity of the shoulder and hand symptoms conferred by a special term may stimulate more frequent and earlier diagnosis as well as more rational, less strenuous therapeutic measures than have been reported.

The classic concept of reflex neurovascular dystrophy following some form of external violence has been established so firmly by Sudeck's description of post-traumatic osteoporosis that a history of trauma has come to be expected or assumed when trophic symptoms are encountered in an extremity. In medical conditions complicated by reflex dystrophy we are actually confronted with internal irritation or injury as the precipitating factor. It must be concluded, therefore, that the similar clinical pictures seen with the varied etiology consid-

ered here may be produced by either internal or external tissue trauma acting through an identical neurophysiologic mechanism.

A number of seemingly different disorders frequently involving the upper extremity, including the idiopathic shoulder-hand syndrome described in the surgical and medical literature as separate entities appear to be closely related. Although their etiology of precipitating factors varies, these conditions exhibit features which are quite similar and the underlying mechanism seems to be identical, a reflex neurovascular dystrophy. The current neurophysiologic concept of a "vicious circle" mediated through a internuncial pool of active stimuli in the cord provoked and maintained by the primary precipitating conditions explains the mechanism common to all forms of reflex (neurovascular) dystrophy, regardless of etiology. It serves as a useful working basis for the present therapeutic approach.

Treatment by sympathetic interruption (with stellate and upper dorsal ganglion block or surgery) is effective in a great number of all etiologic varieties, in that way confirming the common identity of the underlying mechanism.

An excellent discussion of the clinical and roentgenographic changes seen in this syndrome following myocardial infarction, post-hemiplegic and post-herpetic cases is contained in the original article.—*Eugene J. McDonald, M.D.*

Gilbert, Rene, and Voluter, Georges. Contribution à l'étude radiologique des modifications osseuses et cutanées concomitantes dans la région des jambes. (Contribution to the roentgen study of concurrent skin and bone changes in the leg.) *Acta radiol.*, 1948, *29*, 403–428.

In varicose ulcer, changes in the bone structure and periosteal reactions may be seen in the bones of the leg before cutaneous changes are detectable. The authors have shown that osseous changes in the unaffected leg were noted in cases of varicose veins without ulcer. In their opinion the disorganization of the venous circulation, apparent only on one side, may give rise to osseous changes in the opposite leg (which presents no clinical signs).

They conclude that the varicose syndrome, unilateral in appearance, is in reality a bilateral process in which dystrophic disturbances may involve the skin and other soft tissues without being recognizable. These alterations may be associated with osseous changes.—*Mary Frances Vastine, M.D.*

Highsmith, LaRue, S., and Phalen, George S. Sideswipe fractures. *Arch. Surg.*, May, 1946, *52*, 513–522.

The term "sideswipe" is applied to a mutilating fracture of the left elbow, since it denotes the manner in which it was sustained. The driver of an automobile has his left arm resting on the window frame, with his elbow projecting from the window, when his car is sideswiped by an oncoming vehicle or struck by some overhanging projection. Because of the mode of occurrence of this particular fracture, it has also been aptly termed a "car window elbow," a "driver's seat fracture," or a "traffic elbow."

The authors believe that the occurrence of this fracture could be largely prevented if the medical profession as well as the general public could be better informed concerning the manner in which the fracture is sustained. Seven cases are presented and the treatment is discussed in detail.—*James J. McCort, M.D.*

Lyford, John, III, Johnson, Robert W., Jr., Blackman, Sam, and Scott, Roger B. Pathologic findings in a fatal case of disseminated granuloma inguinale with miliary bone and joint involvement. *Bull. Johns Hopkins Hosp.*, Nov., 1946, *79*, 349–357.

The pathologic findings in a fatal case of granuloma inguinale are detailed. The foci infected by the Donovan bodies consist of great numbers of closely packed mononuclear cells which are foamy in appearance and are evidently full of lipoid. The lymph nodes are completely destroyed and replaced by these cells where they grow to form elongated narrow cells which form criss-crossing bundles almost like a fibrous tissue tumor. In the liver, spleen, bone marrow and bronchial lymph nodes there are miliary abscesses which contain some of these cells and other pyknotic necrotic cells and nuclei. The endometrium, fallopian tubes and ovaries are extraordinarily infected and contain myriads of these foamy cells. In sections of tissues stained with Giemsa stain there appear in the cytoplasm of the foamy cells, granular, bluish stained forms corresponding to the Donovan bodies. These findings are a further demonstration that granuloma inguinale is a general systemic disease which does involve bone, joints

soft tissues, internal organs and can terminate fatally.—*James J. McCort, M.D.*

OOSTHUIZEN, S. F., and BARNETSON, JAMES. Two cases of lipomatosis involving bone. *Brit. J. Radiol.*, Oct., 1947, *20*, 426–432.

Two cases are presented. Each was an adult male with a history of some enlargement of the right foot at birth. One patient was a white European, the other a native South African. Each had progressive enlargement of the foot, and after complete roentgenological and both gross and microscopical pathological examination it was found that each had large deposits of adult type adipose tissue in and about the bony structures of the foot, in some areas replacing or destroying bone. In both there was overgrowth and enlargement of the bones involved with distortion and destruction of some portions of bones and expansion of other areas. Most of the joints had been obliterated by the process and there were large lobulated soft tissue tumors. In both patients there was localization to the foot.

No similar case could be found in the available literature.—*E. F. Lang, M.D.*

SHALLOW, THOMAS A., and WAGNER, FREDERICK B. Pulsating benign giant cell tumors of bone; report of a case and review of the literature. *Arch. Surg.*, June, 1946, *52*, 661–676.

A case of pulsating benign giant cell tumor of bone is presented in detail and 6 additional cases from the literature since 1900 are reviewed. The histopathologic picture is indistinguishable from the ordinary variety of giant cell tumor. The pulsation probably depends on free communication of the capillary network of the tumor with its arteriolar and arterial supply, looseness of the supportive stroma and erosion of the bony cortex.

This tumor must be differentiated from other pulsating tumors of bone, namely telangiectatic osteogenic sarcoma, metastatic carcinoma from the thyroid or kidney, and benign and malignant hemangiomatous tumors.

Treatment should consist in wide surgical resection of involved bone whenever possible. If this procedure is impractical, curettage supplemented by radiation therapy may be employed. —*James J. McCort, M.D.*

MCMURRAY, BARRY. A report of six cases of coxa magna following synovitis of the hip joint. *Brit. J. Radiol.*, Nov., 1947, *20*, 477–481.

Enlargement of the femoral head occurs in congenital subluxation, pseudocoxalgia, occasionally following suppurative or tuberculous arthritis, and after synovitis. Of the patients in the series, all had suffered a fairly severe infection for at least five weeks; this was followed by a limp. There was enlargement of the corresponding inguinal glands and palpable and periarticular thickening. Because of the prolonged course of the disease and lack of advancing roentgen changes, the diagnosis of tuberculous arthritis was made in each, but later this was questioned because of rapid response to rest. Enlargement of the femoral head began in 2 of the patients within six months of the onset of the disease, and in the other 4 only after ten months. All were discharged from the hospital within eighteen months after admission and were symptom free. Four patients, seen three, four, ten and fourteen years later respectively, were symptom free. Two others, seen six and eight years later, had developing osteoarthritis.—*E. F. Lang, M.D.*

B. R. KIRKLIN

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